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H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.

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PRESIDENT FERGUSON HAS HEAVY SCHEDULE

President Ferguson is maintaining the fast pace of recent past-presidents of the A. V. M. A. in getting around to veterinary meetings in different parts of the country. In October, he attended the annual meeting of the Eastern Iowa Veterinary Association, at Cedar Rapids. The past month, he went to Lafayette, Indiana, to participate in the Veterinary Short Course held at Purdue University. This month, he will attend the meetings, in Chicago, of the Illinois State Veterinary Medical Association and the United States Live Stock Sanitary Association. In January, he will go to Manhattan, Kansas, to attend the joint meeting of the Kansas State Veterinary Medical Association and the Kansas State Agricultural College Veterinary Conference. The third week in January, he is scheduled to attend the Cornell University Veterinary Conference, at Ithaca, New York, as well as the annual meeting of the Indiana State Veterinary Medical Association, in Indianapolis. Later in the month, he will go to Blacksburg, Virginia, where he will be on the program of the Veterinary Short Course at the Virginia Polytechnic Institute.

The early part of February will see Dr. Ferguson in the South, to take part in the Veterinary Short Course at Alabama Polytechnic Institute. In March, he will go to Columbus, Ohio, where he has agreed to take part in the Veterinary Conference at the Ohio State University. In June, President Ferguson will

attend the joint meeting of the two Carolina associations, at Charlotte, North Carolina, as well as the annual meeting of the Michigan State Veterinary Medical Association, at East Lansing. Whether it will be possible for Dr. Ferguson to accept any additional invitations is doubtful. There is a limit to the time that a busy practitioner can take away from his practice. Also, there is a limit to the appropriation which is available to the President for traveling expenses. It is just about impossible to get more than twelve out of a dozen.

Incidentally it might be mentioned that A. V. M. A. officers have attended 84 veterinary meetings during the past seven years, an average of twelve a year.

DOCTOR WELCH NOW STATE VETERINARIAN

Dr. W. H. Welch, of Lexington, Ill., has been appointed State Veterinarian of Illinois by Governor Emmerson, succeeding Dr. F. A. Laird, who held the same office for a number of years. Dr. Welch is very well known to the profession throughout the United States, having been President of the American Veterinary Medical Association for the year 1922-23. For the last few years, he has been Secretary of the Illinois State Veterinary Medical Association. At the present time he is chairman of the A. V. M. A. Committee on Policy. We are very glad indeed to see a man with the qualifications possessed by Dr. Welch appointed to this very important position.

Dr. Welch was the guest of honor at a dinner tendered him by the McLean County (Ill.) Veterinary Medical Association, at Bloomington, November 8. About fifty veterinarians and friends of Dr. Welch attended.

GRASPING AN OPPORTUNITY

For several years the A. V. M. A. has been affiliated with the American Association for the Advancement of Science. The A. V. M. A. is assigned to Section N, which includes the medical sciences. On frequent occasions members of the A. V. M. A. have contributed to the programs of annual meetings of the A. A. A. S. This year, however, at the meeting in Des Moines, we will see quite an innovation as far as the A. V. M. A. is concerned. Here is how it came about.

Early in the year the A. V. M. A. Representative to the A. A. A. S., Dr. Ward Giltner, received an invitation for the

A. V. M. A. to take a rather prominent part in the program for Section N at the Des Moines meeting, which will be held during the Christmas holidays. The proposal was very carefully considered by the Executive Board at the Detroit meeting and a recommendation was made to the Association that we accept the invitation and that the President be authorized to appoint a committee to assist Dr. Giltner in arranging the program.

President Ferguson appointed the Committee: Dr. Ward Giltner, Chairman, Dr. C. H. Stange and Dr. C. P. Fitch. The following tentative program has been announced for the session to be held Monday, December 31:

Animal Diseases—A Menace to the Human Family—Dr. John R. Mohler, Chief, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Salmon Poisoning—Dr. B. T. Simms and Dr. J. N. Shaw, Oregon State Agricultural College, Corvallis, Ore.

The Relations of Medical and Veterinary Parasitology—Dr. Maurice C. Hall, Chief, Zoological Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Experimental Tuberculosis by the Intracerebral Method of Inoculation—Dr. Wm. H. Feldman, The Mayo Foundation, Rochester, Minn.

Veterinary Medicine and Public Health—Dr. P. A. Fish, Dean, New York State Veterinary College, Cornell University, Ithaca, N. Y.

Progress in Veterinary Education—Dr. C. H. Stange, Dean, Division of Veterinary Medicine, Iowa State College, Ames, Iowa.

The Spread of *Bacterium Abortus* Bang by the Vaginal Discharges of Infected Cattle—Dr. C. P. Fitch and Dr. A. L. Delez, University of Minnesota, St. Paul, Minn.

Brucellosis (Undulant Fever) in Northern Africa and Southern Europe—Dr. I. F. Huddleson, Special Expert of U. S. Public Health Service, Washington, D. C., and Research Associate, Michigan Agricultural Experiment Station, East Lansing, Mich.

The Epidemiology and Prevention of Undulant Fever in the United States—Dr. A. V. Hardy, Director, State Hygiene Laboratories, U. S. Public Health Service, Iowa City, Iowa.

Some Aspects of the Sociologic, Biologic and Ecomonic Phases of the Tuberculosis Problem—Dr. A. F. Schalk, North Dakota Agricultural College, Fargo, N. D.

The Development of an Effective Prophylactic for Dog Distemper—Dr. H. K. Wright, delegated representative of the A. V. M. A. to investigate "Field Distemper Research in England," Philadelphia, Pa.

Technical Supervision in the Production of Highest Quality Milk—Dr. J. G. Hardenbergh, Walker-Gordon Laboratory, Plainsboro, N. J.

APPLICATIONS FOR MEMBERSHIP

(See July, 1929, JOURNAL)

FIRST LISTING

ASHMORE, C. DELMER
209 Livestock Exchange, Union Stock Yds., Los Angeles, Calif.
D. V. S., U. S. College of Veterinary Surgeons, 1910
Vouchers: L. F. Conti and L. M. Hurt.

BETT, THOMAS PATTULLO 12041 E. Jefferson Ave., Detroit, Mich.
B. V. Sc., Ontario Veterinary College, 1929
Vouchers: F. D. Egan and R. K. O'Neil.

COBBETT, NORMAN G. 521 N. Cherry St., Galesburg, Ill.
D. V. M., Colorado Agricultural College, 1921
Vouchers: Z. C. Boyd and J. Stotchik.

HOUSER, ROY Bourbon, Ind.
D. V. M., Grand Rapids Veterinary College, 1918
Vouchers: F. C. Tucker and R. A. Craig.

KIFF, WALTER J. 328½ Franklin St., Michigan City, Ind.
D. V. M., McKillip Veterinary College, 1916
Vouchers: J. C. Schoenlaub and J. P. Ryan.

MAGRANE, HARRY J. 1207 Lincoln Way, West, Mishawaka, Ind.
D. V. M., McKillip Veterinary College, 1913
Vouchers: F. C. Tucker and J. C. Schoenlaub.

MATHEWS, FRANK P. 204 S. Salisbury, West Lafayette, Ind.
D. V. M., Colorado Agricultural College, 1923
Vouchers: John F. Bullard and R. A. Craig.

STRIGGLE, CLAUDE WALTER South Whitley, Ind.
D. V. M., Indiana Veterinary College, 1915
Vouchers: Harvey E. Whiffing and J. P. Ryan.

WARREN, DAYTON MCRAE 529 N. Rodney St., Helena, Mont.
D. V. M., Ohio State University, 1924
Vouchers: Hadleigh Marsh and E. D. Nash.

Applications Pending

SECOND LISTING

Blackman, Raymond A., Box 212, Vienna Va.
Farley, Herman, Dept. of Vet. Path., Kansas State Agr. College, Manhattan, Kans.
Gonzaga, Arcadio C., College of Vet. Science, Agr. College, Laguna, P. I.
Grefsheim, Thorval, Deerfield, Wis.
Hays, Mack, Box 2246, Alabama Polytechnic Institute, Auburn, Ala.
McCoy, James Owen, Reeseville, Wis.
Mackey, Graham L., Bedford, Va.
Meyer, E. Albert, 2958 Elizabeth St., Denver, Colo.
Mott, Lawrence Orville, Vet. Hospital, Kansas State Agr. College, Manhattan, Kans.
Schwarte, L. H., Vet. Investigation, Iowa State College, Ames, Iowa
Swingley, Lee B., 700 Jackson St., Oregon, Ill.
Williamson, Charles O., Powell, Wyo.
Zimmerman, Israel, 16 Poplar Terrace, Bridgeport, Conn.

The amount which should accompany an application this month is $5.42, which covers membership fee and dues to January 1, 1930 including subscription to the JOURNAL. It is suggested that applications filed this month be accompanied by remittance for $10.42, the additional $5.00 being for the 1930 dues.

COMING VETERINARY MEETINGS

Horse Association of America. Palmer House, Chicago, Ill. December 4, 1929. Mr. Wayne Dinsmore, Secretary, Union Stock Yards, Chicago, Ill.

New York City, Veterinary Medical Association of. Academy of Medicine, 103rd St. & 5th Ave., New York, N. Y. December 4, 1929. Dr. Raymond J. Garbutt, Secretary, 305 W. 91st St., New York, N. Y.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. December 4, 1929. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

U. S. Live Stock Sanitary Association. La Salle Hotel, Chicago, Ill. December 4-6, 1929. Dr. O. E. Dyson, Secretary, 45 Live Stock Exchange Bldg., Wichita, Kans.

National Association of B. A. I. Veterinarians. La Salle Hotel, Chicago, Ill. December 4-6, 1929. Dr. J. S. Grove, Secretary, 1715 Belmont Ave., Fort Worth, Texas.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. December 10, 1929. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.

Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. December 10, 1929. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

Nebraska State Veterinary Medical Association. Grand Island, Nebr. December 10-11, 1929. Dr. B. Witt, Secretary, Scribner, Nebr.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. December 11, 1929. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

Western New York Veterinary Medical Association. Erie County Society for the Prevention of Cruelty to Animals Building, Buffalo, N. Y. December 12, 1929. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. December 18, 1929. Dr. W. L. Curtis, Secretary, 1264 W. Second St., Los Angeles, Calif.

American Association for the Advancement of Science. Des Moines, Iowa. December 27, 1929-January 2, 1930. Dr. Burton E. Livingston, Permanent Secretary, Smithsonian Institution Bldg., Washington, D. C.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 6-10, 1930. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Kansas State Veterinary Medical Association and Kansas State Agricultural College Conference for Veterinarians. Kansas State Agricultural College, Manhattan, Kans. January 7-8, 1930. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans.

University of Pennsylvania, Conference of Veterinarians at. School of Veterinary Medicine, University of Pennsylvania,

Philadelphia, Pa. January 8-9, 1930. Dr. Louis A. Klein, Dean, 39th St. & Woodland Ave., Philadelphia, Pa.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 8-9, 1930. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Oklahoma State Veterinary Medical Association. Huckins Hotel, Oklahoma City, Okla. January 13-14, 1930. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 14-15, 1930. Dr. Cecil Elder, Secretary, University of Wyoming, Laramie, Wyo.

Wisconsin Veterinary Medical Association. Madison, Wis. January 14-16, 1930. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Iowa Veterinary Medical Association. Ft. Des Moines Hotel, Des Moines, Iowa. January 14-17, 1930. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Minnesota State Veterinary Medical Association. Hotel Lowry, St. Paul, Minn. January 16-17, 1930. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

Cornell University, Annual Conference for Veterinarians at. Cornell University, Ithaca, N. Y. January 16-17, 1930. Dr. P. A. Fish, Dean, New York State Veterinary College, Ithaca, N. Y.

South Dakota Veterinary Medical Association. Hotel Cateract, Sioux Falls, S. Dak. January 16-17, 1930. Dr. W. P. Brower, Secretary, Canton, S. Dak.

Indiana Veterinary Medical Association. Hotel Severin, Indianapolis, Ind. January 21-23, 1930. Dr. R. H. Boyd, Secretary. 1422 N. Capitol Avenue., Indianapolis, Ind.

Nevada State Veterinary Association. Reno, Nevada. January 22, 1930. Dr. Edward Records, Secretary, University of Nevada, Reno, Nevada.

Virginia Polytechnic Institute Veterinary Short Course. Blacksburg, Va. January 27-31, 1930. Dr. Russell A. Runnells, Virginia Polytechnic Institute, Blacksburg, Va.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 28-31, 1930. Dr. Ward Giltner, Dean, Division of Veterinary Science, Michigan State College, East Lansing, Mich.

Delaware Veterinary Medical Association. Dover, Del. January 29, 1930. Dr. J. R. Porteus, Secretary, Box 365, Smyrna, Del.

PUBLIC HEALTH ASPECTS OF UNDULANT FEVER*

By WALTER W. LEE, *Indianapolis, Ind.*
Assistant Secretary, and Epidemiologist
Indiana State Board of Health

Until the last three years undulant fever, or Malta fever, as it was better known, was but one of the medical curiosities. Occasionally an immigrant from the Mediterranean area would develop an undulating fever and drift into our hospitals. But recently this disease has leaped into the limelight and, with tularemia, for the time being at least, holds the center of the stage in veterinary, medical and public health interest in North America.

The etiological agent was discovered by Bruce, of the British Army, in 1887, when he was studying Malta fever in the island of Malta. He established *Micrococcus melitensis* as the common factor in both the human and goat diseases. In 1897, Bang established *Bacillus abortus* as the etiological factor in contagious abortion in cattle. In 1912, Schroeder and Cotton found Bang's bacillus in milk of aborting cows, and at that time anticipated the possibility of human infection from the use of such milk in the raw state. In 1918, Evans found a very close relationship between *Br. melitensis* and Bang's bacillus,[1] as close as is the relationship between the various groups of meningococci. In fact, they can be differentiated definitely only by agglutinin absorption. Evans also reclassified the melitensis groups on a basis of agglutinin reactions. Those prevalent in America she classified as *Br. melitensis* var. *melitensis* or the caprine and human strain, and *Br. melitensis* var. *abortus* of bovine or porcine origin. She found also that the type of organism was not necessarily related to the last animal host. She had one melitensis variety of the goat type from a mare in Iowa, and, since, the same type has been isolated from a cow in Alexandria, Va. Huddleson reports two cultures isolated from two mares, one a bovine, the other a porcine strain. Cattle are frequently infected by the porcine strain, and hogs with the bovine strain. Man may likewise be infected by all three strains, but recent findings seem to indicate that the bovine strain is less virulent for man than are the others.

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

In 1923, Evans reported the inoculation of a pregnant heifer with *Br. melitensis*, with subsequent abortion and the isolation of the same strain from the fetus. Early in 1924, Keefer reported a case of undulant fever in man at Baltimore, definitely proven to be of bovine origin. This is the first case of its kind on record. Following this case other cases of human infection by the bovine organism were reported by Evans, Carpenter, Huddleson, and others. By 1927 the laboratories of many of the state boards of health were searching for and finding human infections, until now over 1,000 cases have been reported from practically all over the United States and Canada. One might safely add that it is being found in direct proportion to the effort expended in looking for it.

Widal Samples Tested for Malta Fever

In Indiana the laboratory began, in 1927, to examine all bloods sent for Widal tests, for agglutination against *Br. melitensis*, and found one in less than three weeks. The patient was an eighteen-year-old boy attending school at Valparaiso, Indiana. He was taken ill with a fever, December 26, 1926. He apparently recovered in a few days, returned to school, coming down early in January, 1927, again with an acute fever simulating typhoid fever. Blood was sent by his physician to eliminate typhoid fever from the diagnosis. It was found to be negative for typhoid, but positive for *Br. melitensis* in a dilution of 1:640. The patient recovered early in February, 1927. His physical examination was indefinite except for fever, the symptoms being those of toxemia. The blood was cultured, but the organism was not found. In December, 1927, this patient's blood still agglutinated *Br. melitensis* completely in 1:270, and partially in 1:540. King, of the Mt. McGregor (N. Y.), Sanatorium, found by agglutinin absorption that the infection was of the bovine strain.

In September, 1927, an outbreak of undulant fever occurred in South Bend, Indiana,[2] which was traced definitely to a herd of cows infected with contagious abortion. South Bend had shortly before passed a milk ordinance which required pasteurization of all milk not from tuberculin-tested cows. After this epidemic the city fathers responded to the arguments of the local health officer and amended their ordinance, requiring complete pasteurization of all milk sold in South Bend. The city is to be congratulated upon its epidemic, for, because of it, the city won its pasteurization ordinance. South Bend citizens owe

Br. melitensis a deep debt of gratitude, for it has, no doubt, made impossible for all time milk-borne epidemics of worse diseases than undulant fever.

In 1928, two cases developed in Fort Wayne. Both individuals drank raw milk. One was a physician and one a laborer. The herd supplying the milk used by the physician was found to be infected with contagious abortion. The council of Fort Wayne considered but failed to pass an ordinance prohibiting the sale of other than pasteurized milk within the city. In February, 1929, another physician in Decatur, Indiana, who drank milk from the same herd, became ill with undulant fever. Early in 1928, a number of students in an Indiana college[3] became ill with fever simulating typhoid. Examination of the blood of the students showed 14 positive for undulant fever. There were 26 in all with clinical symptoms of undulant fever, 14 positive by agglutinin test, 6 negative for agglutinins, and 6 not tested. The cases were all under the observation of one doctor. An interesting feature of this outbreak is the fact that breeding hogs were not kept on the college farm. There were some fattening hogs, however. Six of these were fed milk from the cows in the herd, which were found to be eliminating *Br. abortus* in the milk. One of these, a sow, gave birth to a litter of pigs. The six fat hogs remained apparently normal and when killed showed no evidence of *Br. abortus* infection, nor was any infection found in the litter of little pigs which were killed and autopsied after birth. Dr. Mathews concludes, from these findings, that the infection in the herd was of the bovine, rather than the porcine, type. *Br. abortus* cultured from the milk of the cows was found by both Mathews and Huddleson to be of the bovine type. Blood cultures of the patients were all sterile.

Ordinances Prohibit Sale of Raw Milk

As in South Bend, the city of Richmond, Indiana, profited by its epidemic, and the city council passed an ordinance prohibiting the sale of raw milk in the city of Richmond.

Altogether, some 60 cases of undulant fever have been found in Indiana since January, 1927. It might be of interest to note, in passing, that 4 of the 60 patients were physicians. Physicians all know, or should know, the danger of drinking unpasteurized milk. One can imagine only one thing more ridiculous than for a physician to contract undulant fever, and that is for a health officer to get it. New York State reports a health officer-physician

who preferred raw to pasteurized milk and recently contracted undulant fever. When professional health men will not adopt the universally recognized precautions against milk-borne disease, the prospect for its control among the laity is dark indeed. These sixty cases of undulant fever, which have been diagnosed in Indiana, do not by any means represent the extent of the infection in the State. Giordano[4] reports, out of 1100 routine sera, 63 positives, among which 14 are clinically positive. These are all from the neighborhood of South Bend. Two cases were found in Fort Wayne and one in Decatur, near by; one in Michican City, one in Goshen, one at Valparaiso and twenty-eight cases, all infected by one herd, in Richmond, Ind. Over half of the cases in Indiana were found in the northern tier of counties, where there are two efficient private laboratories, which found all but two of the cases in that region. All of the rest are practically one epidemic, all under the supervision of one physician. There is no reason to think that contagious abortion is not evenly distributed throughout the herds of the State. The laboratory of the State Board of Health is running bloods sent for Widal tests and also Wassermann sera but to date have found only two positive. Without a doubt, if cases of undulant fever were looked for by private physicians, and blood sent from the cases to the laboratory, many more cases would be found in all parts of the State.

Clinical Symptoms not Characteristic

The clinical symptoms of undulant fever are not of themselves sufficiently characteristic upon which to establish a diagnosis. The most characteristic symptom is that of agglutination of the specific organism by the serum of the patient. Positive laboratory findings together with clinical evidence establish the diagnosis. Either one or the other may be absent. In the Richmond, Ind., outbreak, six cases with clinical symptoms had no agglutinins in their blood, and cases are frequently found where the sera agglutinate the organism but clinical evidence is absent. The titer of the serum reaction is not of importance in diagnosing the disease. Cases with obvious clinical symptoms have been reported showing titers as low as 1:10. Some sera will agglutinate in high dilution, but not in low dilution. McAlpine and Mickle found four sera, out of over 10,000, which agglutinated *Br. melitensis* completely in a dilution of 1:200, or higher, but not below. Francis has found that there is cross-agglutination

between tularemia and melitensis which must be borne in mind. Some men use a polyvalent antigen, including several different strains of the *Br. melitensis* groups. Giordano claims to have found cases reacting to one strain and not to others. Other laboratories use but a single strain antigen.

There are still a great many details to be worked out with reference to this disease. We still need to know more about the spread of the organism and its infection of the host. We know that infected guinea pigs manifest the disease by toxemia symptoms during life, and involvement of the lymphatics, liver and spleen not unlike tuberculosis. In fact, a few years ago, instances of apparent tubercular involvement of liver and spleen in guinea pigs would prove sterile on culture. Perhaps these were cases of melitensis infection. Cattle usually manifest the infection by abortion, and the organisms are found in abundance in the fetus, fetal membranes, uterine discharges and often in the milk. The presence of *Br. melitensis* in the milk may or may not coexist with mastitis. The bull may develop an orchitis. In man the symptoms of infection are often indefinite. The chief symptom is a more or less prolonged fever with symptoms of toxemia. It may simulate, therefore, typhoid fever, tuberculosis, endocarditis, malaria or influenza. Some cases have manifested arthritic symptoms, and, therefore, have been confused with acute rheumatic fever or tuberculosis of the bone. Since abortion is so common a symptom in animals, it would be expected to occur likewise in the human host. Few cases of such have been reported. It is obvious that this disease should be suspected where human abortion occurs without other obvious cause; but, as yet, this has not proved to be so common a symptom as might be expected.

Few Children Reported Affected

Another peculiarity of this infection deserves study. Although children are the greatest consumers of milk, exceedingly few cases, some two or three, have been reported as occurring under five years of age. This may mean either that children are not susceptible to undulant fever, or that it may be manifesting itself in a form not yet recognized as such. Young cattle appear to be resistant to this infection before they reach the age of sexual maturity. Some veterinarians say it is quite safe to feed calves milk from infected cows, but authorities are tending to look upon such a practice with suspicion, and have registered their dis-

approval. They are not sure that calves may not acquire the infection and retain it in a more or less latent or dormant form, to become active later on. As yet, no one has explained satisfactorily the apparent lack of susceptibility of the young of animals or humans to undulant fever.

Although the usual mode of human infection is by way of the gastro-intestinal tract through the drinking of milk contaminated with the organism, several cases have developed accidentally among laboratory workers who have been working with the organism. Most laboratories which have done much work with *Br. melitensis* have had one or more accidental infections among their staff. The caprine strain seems to be the most dangerous to laboratory workers, and is particularly so when used in animal experimentation. Although laboratory workers are very prone to contract undulant fever from experiment animals, apparently by contact, the writer has not read of any cases of undulant fever transmitted from a human case to attendant by contact.

Incidence of Malta Fever

It is impossible as yet more than to guess at the probable incidence of undulant fever in the country. Efforts have been made to get a cross-section of the population by testing sera sent to laboratories, for routine Wassermann tests, against *Br. melitensis*. Evans[1] examined 500 sera and found 50 (11.8 per cent) positive in dilutions of 1:5 and higher, one of which was 1:320, the only one above 1:40. This patient appeared well. Carpenter[5] and Chapman found 7.3 per cent of positives in 4050 routine Wassermann bloods in Syracuse. McAlpine and Mickle[6] found, out of 20,259 routine Wassermann sera, 127 positive, in dilutions of 1:100 and over. Of these, only 20 showed clinical symptoms of undulant fever.

The age and sex distribution of this disease is of interest. All ages over five years are affected, but the greatest incidence occurs in young adults to middle life, with the males affected about three times as frequently as females. Hardy[8] found, out of 125 clinical cases in Iowa, 98 were males and 27 females. Sixty-eight per cent of the male cases developed between the ages of 20 to 44. The female cases were likewise distributed from 5 to 75 years of age but with only slightly greater incidence during adult life.

In investigations, where cases are found by examining random sera, and where clinical symptoms are absent, the laboratory

positives seem to be rather equally distributed among the sexes. The data to date are insufficient upon which to establish an opinion but perhaps future findings may show that both sexes are being equally infected, but the male sex is more apt to develop the disease with clinical manifestations.

Spread by Contaminated Milk

There can be no doubt at the present time that undulant fever is transmitted from cattle to man by contaminated milk from infected cows. But although this is true, we also know that few persons who drink such milk contract the disease. The factor of resistance of the exposed individual enters into the equation and is quite as important a factor as exposure. This same phenomenon is observed every day in other diseases, such as typhoid fever. In a recent outbreak of typhoid fever in Fort Wayne, when untreated river water was accidentally pumped into the city mains, several thousand persons became suddenly ill with gastro-enteritis, but only 53 persons out of all those exposed developed typhoid fever. Apparently typhoid fever infection is a relatively rare accident when one takes into consideration the amount of exposure in the entire population. Likewise, in the case of undulant fever, we see thousands of people drinking unpasteurized milk from cows known to have aborted, yet only now and then will an individual develop symptoms of undulant fever. True, relatively few physicians are looking for this disease, and many cases are, no doubt, being diagnosed as something else; yet this same condition holds where physicians are looking for this particular infection. Orr and Huddleson[7] examined the blood from 250 males and 250 females out of 800 individuals in an asylum for epileptics. These persons were using raw milk from a herd of cows known to be eliminating *Br. abortus* in their milk. Out of this group only seven persons showed agglutinins in dilutions of from 1:25 to 1:500, and only one of these seven showed clinical symptoms. The degree of infection in this group with known massive exposure was practically identical with that found by McAlpine and Mickle[6] in Connecticut, by examining routine Wassermanns taken at random from the population. Several men have fed volunteers pure cultures of *Br. abortus*, of both bovine and porcine strains, with negative results.

The following experience is of especial interest, and is quoted from a paper by King and Carpenter.[5]

Dr. King observed a case of undulant fever at the Metropolitan Life Insurance Company's sanatorium at Mt. McGregor, N. Y. This prompted an investigation of abortion disease in the sanatorium herd, as well as a routine examination of the serum from all patients and the staff at this institution. The herd consisted, at this time, of 150 cows. A blood test of this group showed 82, (54.6 per cent) of the animals to be infected with *Br. abortus*. The milk from the infected animals was then examined and 24 (29.2 per cent) of them were found to be eliminating the organism.

A routine test on the serum from the 530 patients and staff showed 69 (13 per cent) to have abortus agglutinins. Eight of these had shown definite symptoms of undulant fever. Many others had a history of symptoms suggestive of the milder form of the disease. All of the patients had been drinking large amounts of the raw milk and cream from this herd. Because of these findings the infected cows were segregated, and the milk from the cows discharging abortus was pasteurized. We then considered the milk supply to be free from living abortus infection. The routine serum examinations on 448 new patients and employees was continued after the milk was supposedly abortus free. The results of these tests have shown only 6 of the 448 to have a few abortus agglutinins. The 6 serums produced only sedimentation of the antigen when the serum was diluted 1:15. At the time we observed these 6 serums we discovered that one cow, whose milk had been negative repeatedly, began to discharge *Br. abortus* after a normal gestation period. Unknowingly, her infected milk was mixed with the raw abortus-free milk for approximately 6 weeks before it was detected. The examination of serums for approximately 100 patients for a period of 3 months since the pasteurization of her milk has given negative results.

Of this group of 530 individuals, all of whom drank large quantities of this infected milk, only 13 per cent showed agglutinins, and but 1.5 per cent of the group developed the disease in clinical form. Of a new sanatorium population drinking the milk from the same herd, but with the reacting cattle segregated and their milk pasteurized, only 1.3 per cent showed agglutinins in their blood and that was apparently due to the introduction of the milk of one infected cow into the raw milk of the herd, before she was detected. This furnishes an object lesson which all men interested in public health would do well to consider. If in a herd, owned by the Metropolitan Life Insurance Company, under constant veterinary and laboratory supervision, and being constantly studied with reference to undulant fever, one cow could be discharging *Br. abortus* in her milk for six weeks before being detected, what hope have we of producing safe raw milk even though certified? Carpenter found 6.08 per cent of 378 cows producing certified milk eliminating *Br. abortus* in their milk.

It is quite true that in the past no effort has been made to eliminate the aborting cows from the herds producing raw milk, because we did not suspect that the infection could be transmitted from cattle to man. In the future, no doubt, such infected cows will be rejected, but the experience of the Metropolitan Life Insurance Company's sanatorium and other findings show

that constant vigilance will be necessary to weed out infected cows, which, to be adequate, would require technic financially impossible for even certified dairies, much less the ordinary dairy producing raw milk.

Although milk is the chief medium for the transmission of undulant fever from animals to man, milk products also may carry it. Cream carries the organisms in greater concentration than the milk, because the organisms tend to rise with the cream when the milk is allowed to stand. Carpenter and Boak[9] found that in cream artificially inoculated with *Br. abortus* and stored at 8° C., the organisms remained viable for eight to ten days. *Br. abortus* remained viable when inoculated into butter made from sweet unpasteurized cream, and stored at 8° C. up to 142 days. His opinion is that market butter made from sour cream and cheese rarely, if ever, carry the disease. The deciding factor in the viability of *Br. abortus* in milk and its products seems to be the hydrogen-ion concentration. An acidity greater than pH5 destroys the organism.

Malta Fever of Major Importance

Although it appears at the present time that infection of human beings with undulant fever is a relatively rare accident compared with the degree and frequency of exposure, yet it occurs with sufficient frequency that this disease is one of our major public health problems, probably as important as typhoid fever.

There is another factor which we must consider. Evans[10] has a caprine strain of *Br. melitensis* isolated from a mare in Iowa, one from a cow in Maryland and one from a cow in Alexandria, Va. She also reports cases of men infected by the caprine strain in Alexandria, Va., and in North Carolina. The caprine strain of *Br. melitensis* is the particular type of organism causing the Mediterranean type of undulant fever commonly known as Malta fever, and is much more virulent for man than either the bovine or porcine strain of the abortus organism. Some two score of cases of the true Malta fever[11] caused by the caprine strain of *Br. melitensis* have been reported from the southwestern states since 1922. These were all traced to herds of goats which were originally imported from the Mediterranean area. The patients were all users of raw goat milk. With the caprine strain of *Br. melitensis* infecting goats in the United States, and known instances of the same infection in cattle, the stage is apparently set for the spread of the more virulent type of this disease through

our cattle. Should the caprine strain ever become established in the cattle in this country, undulant fever will become an epidemic disease of first magnitude from the standpoint of public health.

Fortunately we have had the preventive since before we knew we had the disease, so that our problem now is to educate the public to avail themselves of the preventive, which is pasteurization of all milk and cream used for human food. Park has found the thermal death point for this organism to be 140° F. for fifteen minutes. This finding is well established, so the ordinary pasteurization temperature at 145° F. for thirty minutes is more than sufficient to destroy all abortus infection in milk.

Prevention is Simple Matter

The prevention of undulant fever in human beings is simplicity itself, compared with the problem of the farmer and veterinarian in eliminating the disease among the cattle.

Undulant fever is a disease with a low mortality but with a prolonged and indefinite period of recovery when it succeeds in establishing itself in man. No person need have undulant fever if he will but avail himself of the means of prevention, namely, use no milk that has not been pasteurized. Bigelow and White[12] report more than 50 milk-borne epidemics which have been recorded, involving some 30,000 cases of disease. From 1911 to 1928 inclusive, there have been, in Massachusetts alone, 1468 cases of typhoid fever, 74 of diphtheria, 1147 of scarlet fever and 3986 of septic sore throat attributed to milk as a carrier.

A few years ago the San José scale and other insect pests threatened to destroy our fruit crops. Scientific agriculture introduced poison sprays and clean and sane cultural methods, and the fruit-growers not only controlled the insect pests but their agriculture methods were so improved that the increase in crop production resulting from the preventive work more than paid for the cost and inconvenience of pest control. If the fear of undulant fever will hasten the adoption of universal pasteurization of milk, the country will be just that much sooner rid of milk-borne diseases. If the presence of undulant fever will hasten the day when we will have universal pasteurization of milk, then undulant fever may well be looked upon as a benevolent catastrophe, and the world will then owe a debt of gratitude to *Br. melitensis*

SUMMARY

The etiological agent of Malta fever was discovered by Bruce, in 1887, and named *Micrococcus melitensis*. In 1897, Bang found and named *Bacillus abortus* as the cause of contagious abortion in cattle. Schroeder and Cotton, in 1912, found Bang's bacillus in cows milk. Evans established the relationship between the *M. melitensis* and Bang's bacillus and reclassified them on the basis of their agglutinin-absorption qualities. Keefer, in 1924, found the first case of undulant fever due to the bovine strain of *Br. abortus*.

Indiana found its first case in January, 1927. Since then some sixty cases have been found. As a result of outbreaks of undulant fever, two Indiana cities have adopted ordinances requiring pasteurization of all milk within their limits. Clinical symptoms of undulant fever are those of a toxemia and are not sufficiently characteristic upon which to establish a diagnosis. The finding of agglutinins in the blood is of great importance and, when present together with a positive history and clinical findings, establish the diagnosis. The titer of the sera is not of itself of importance in diagnosis. Cases may be positive with titers as low as 1:10. Some sera show positive agglutination in titers above 1:200 and not below. Some laboratories use polyvalent antigen, others use but a single strain antigen. There is cross agglutination between the various strains of *Br. melitensis* and with *B. tularense*. Guinea pigs react to the infection not unlike to tuberculosis, with involvement of the lymphatics, liver and spleen. Cattle manifest the infection chiefly by abortion. In man the disease simulates tuberculosis, endocarditis, malaria, influenza, arthritis, or tuberculosis of the bones. Human abortion is apparently rare as yet. Children do not appear to be susceptible to undulant fever although they consume a large part of the milk used as food. Calves likewise seem to be relatively immune. Accidental laboratory infection is quite common, especially where animal experimentation is done. Although infection is apt to occur by human contact with infected animals, contact infection between human patient and attendant seems to be rare. Examination of random sera would indicate a probable infection of from 1 to 10 per cent of the population, depending upon the dilution at which the sera are examined and the locality. Although laboratory positives are relatively high, clinical cases are relatively rare. Even though clinical cases are relatively rare, undu-

lant fever is probably of equal importance with typhoid fever, from the viewpoint of public health. Data, so far, seem to indicate that clinical cases occur most frequently in male adults.

The experience of the Metropolitan Life Insurance Company's sanatorium, at Mt. McGregor, N. Y., would indicate that the probability that milk can be produced, so that it will be safe for human food, even though certified, without the additional safeguard of pasteurization, is hardly possible. *Br. abortus* will live in cream for eight to ten days at 8° C. and in butter made from sweet cream for 142 days. The viability of this organism seems to depend upon the hydrogen-ion concentration. An acidity greater than pH5 destroys *Br. abortus*. The thermal death point is 140° F. for 15 minutes, so pasteurization is more than sufficient to destroy it in milk. The caprine strain has been found in infected animals and man, in the United States. It is known to be a common infection in goats along the Mexican border. Should this strain become spread among our cattle, the condition would be very serious from the standpoint of public health.

The preventive of undulant fever has been available since before we knew we had the disease, namely pasteurization of milk. If the presence of undulant fever will hasten the adoption of universal pasteurization, the world will then owe a debt of gratitude to *Br. melitensis*, for pasteurization will be the means of preventing the possibility of milk-borne diseases worse than undulant fever.

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DISCUSSION

DR. WARD GILTNER: I greatly rejoice in an opportunity to discuss Dr. Lee's paper. It is a masterly resume of the situation, but no one is in a position to paint the picture of Brucella infection in man or animals and have it hung on the wall permanently. Yet, there are one or two impressions left that I think should be corrected or at least a warning should be issued.

I doubt very much whether the melitensis or caprine strain is the most serious of the strains, or species, as we call them, of this organism. The work of Huddleson indicates almost conclusively that the suis strain is the most serious of the strains in the animal. In work with monkeys it has proved more serious than fresh strains of the melitensis that we could avail ourselves of. Furthermore, in this country I am quite satisfied that, since the suis strain is not uncommon, and since it can invade cattle readily, because of the relation of the two species in the barnyard on the farm, and because we know experimentally that it does attack cattle, we have far less to fear in the future from *Brucella melitensis* than from what we call *Brucella suis*.

Again, from communications received this summer from Dr. Huddleson, who is studying the problem in northern Africa and southern Europe, I get the impression that in southern France the cow is more serious than the goat as far as this disease is concerned. We have no data as to the type of organism that is causing the trouble there. In connection with these communications from Dr. Huddleson, we learn that the problem is certainly very serious around the Mediterranean. Dr. Huddleson just spent ten days in Malta and he encountered a very serious epidemic, I think nearly 200 cases.

This group of organisms is certainly very interesting. A paper just presented a short time ago, in the Section on Poultry by my colleagues, Drs. Emmel and Huddleson, shows that the infection is here in Michigan, at least in four poultry flocks. Just what this type of organism is, I don't know. When we will complete the list of the domesticated and perhaps undomesticated animals, including man, susceptible to this disease, I can't predict, but I do say that this is a very interesting group of organisms.

I am very much in sympathy with Dr. Lee's position relative to pasteurization of milk. Certainly it seems most short-sighted, in fact, quite typically American, not to take advantage of this certain method of controlling this disease in the human family, but I would demur a little. It is possible to produce raw milk, if we have the right kind of control, that is safe as far as these organisms and other pathogens are concerned.

Dr. Torrey, in our laboratory, has perfected a very simple technic which he will demonstrate tomorrow at the Fair Grounds, with the cooperation of Dr. C. H. Case, of Akron, Ohio. I am sure a great many laboratory workers here in the room are using it—a technic whereby you can determine, we feel with great confidence, whether the milk of udders is dangerous. This technic is being used by some of the veterinary laboratory men connected with the herds producing raw milk.

DR. L. A. KLEIN: Mr. Chairman, this disease presents one of the most serious problems of milk control that we have had to deal with for some time. I would like to add to what Dr. Lee has stated in regard to the epidemiology. Dr. Hardy, in his study of some 125 cases in Iowa, found that there was a considerable proportion in which infection could not be traced to milk. In those cases, infection seemed to have been obtained by contact with infected animals, the patients having been in contact with herds in which abortion existed, or with animals or their products in packing houses.

As far as I have been able to learn, while infection has occurred from contact with infected animals among farmers and among packing-house employes, there is only one case on record of a veterinarian having been infected and he was a meat inspector, but I bring this point out because I think it is very important for veterinarians to keep in mind that it is possible for a human being to contract the infection by working with cattle or other animals that have the infection.

I said in the beginning that this was a very difficult problem in milk control. I am not so certain that pasteurization will entirely dispose of all of the difficulties, although pasteurization is a good expedient under certain conditions. The ideal method would be to blood-test dairy herds repeatedly and remove reactors, dispose of them in some manner, and produce milk from cows free from this disease. It has been demonstrated that you can eradicate this disease from dairy herds, and with herds free from this disease we can produce milk free from this infection. Of course, it is not always practicable to use this

method. It can't be applied universally at once, but it can be applied in many herds and it might be introduced gradually. Until that time, of course, pasteurization is the best method we have for dealing with the infection.

But another question to be considered is: what do we actually know about the efficiency of pasteurization in dealing with this infection? It has been demonstrated by laboratory tests that an exposure at 140 degrees for fifteen minutes will kill abortion bacilli of the bovine type. Of course, that would indicate that commercial pasteurization at 140 degrees for 30 minutes will kill the infection, but rather recently in a series of experiments conducted in the New York State Veterinary College at Cornell University it was found that 140 degrees for fifteen minutes did not kill abortion bacilli of the porcine strain, and we know that we may have abortion bacilli of the porcine strain eliminated in the milk of the cow.

We must have more information on the subject, before we can be certain of just what protection pasteurization is going to give. I can't help recalling in this connection that for some years it was assumed that pasteurization of milk at 140 degrees for thirty minutes was an absolute protection against infection with bovine tuberculosis, but we now have a number of cities in this country that require milk to come from tuberculin-tested herds, whether it is to be pasteurized or sold raw, so that I think we should not lose sight of the fact that the ideal method of dealing with this danger is to eradicate the disease from the dairy herds.

Dr. E. M. Pickens: I feel, personally, that pasteurization is undoubtedly one of the greatest instruments that we have for the protection of the public health. At the present time it doesn't seem practical to pasteurize milk in rural communities. It may be later. Inasmuch as it is impossible to have universal pasteurization at this time, wouldn't it be well to spend as much of our energies as possible on methods of obtaining milk from healthy cattle, especially certified herds, as Dr. Klein has pointed out, and not destroy certified milk?

Dr. R. R. Birch: I feel with respect to this precisely as do Dr. Klein and Dr. Pickens. We have a fundamental way to attack the problem—cleaning up our herds—and we have the expedient—pasteurization—to cut down the danger in the meantime.

There are two points in Dr. Lee's paper that I should like to discuss just a moment. I fear a wrong impression may have been left. One is with respect to calves carrying *B. abortus* from calfhood to breeding maturity. That isn't pertinent to the immediate problem but, as a matter of actual fact, it is very rare to find a calf, which has been segregated from mature cattle subsequent to weaning, which reacts to the agglutination test when it reaches breeding maturity.

The other point concerns the undue emphasis placed by Doctor Lee on the very exceptional cow which King and Carpenter had under observation. This cow failed, for a long time, to show agglutinins in the blood, and was, during that time, eliminating *B. abortus* in the milk. In all the examinations we have made we have encountered just one animal of this kind. There are not enough of them to interfere seriously with any plan of eradication.

Dr. C. P. Fitch: Some of the problems that have been presented by Dr. Lee in his paper are some of the most pertinent that pertain to public health at the present time. We have gone through certain methods of control in respect to tuberculosis in so far as it relates to human health, and we are now going through an analogous stage in regard to abortion, and I want to add my word to what has been said.

In my judgment the way to get at this problem is through the herd, building up herds free of this infection which we have demonstrated can be done under relatively easy conditions as you will note in the article which we published in the August number of the Journal of this Association.

Again, it doesn't make any difference, as far as it relates to public health, whether people get the porcine type of organism from the hog or from the cow. The point is that human beings are affected with *Brucella abortus* or *Bacterium abortus*. Whether it is or is not the melitensis, porcine, or the bovine type, I

believe a great deal more will have to be done before we will be able to have a clear picture of these types of organisms presented to us, so that we can say definitely that this organism is and always has been what it is today.

DR. R. C. DAYTON: I would like to know if there is any information at hand regarding the flash pasteurization of milk for the destruction of this organism. You have spoken of the holding method, 140 degrees for fifteen minutes. There are several machines manufactured, I believe, which pasteurize milk at 162, with the flash method, and I think it is well in all of our work not merely to discuss the holding method, but the flash method of pasteurization as well.

DR. GILTNER: Mr. Chairman, may I attempt to answer that question? With the electrical pasteurization, using the apparatus that is available in this country—the only one that I know of—very extensive experiments which Dr. Huddleson and Dr. Carpenter and others have initiated, indicate that there is no question whatever about the efficacy of the flash continuous-flow method of pasteurizing at 160 degrees. They say in that connection that in one case only the abortus resisted heating at 155 degrees, whereas the tubercle bacillus did not. The flash pasteurization with steam is now being developed in Chicago, but I am quite sure that nothing practical has been done with it so far.

May I say also, speaking from Michigan's standpoint, I just don't get Dr. Klein's objection to pasteurization for he talks about 140 degrees for fifteen minutes. One hundred and forty-five degrees for thirty minutes in Michigan's pasteurization, and that is effective as far as abortus is concerned.

CHAIRMAN KELSER: Dr. Lee, will you close the discussion?

DR. LEE: Mr. Chairman and Gentlemen: I see I am in a minority as far as pasteurization is concerned.

In the first place, pasteurization is not an excuse for dirty milk. We should have clean herds and clean milk which should then be made safe by pasteurization.

Dr. Giltner has mentioned a new process of producing milk that will probably be free from undulant fever germs. Anything is possible. Herds may be made reasonably free of tuberculosis by rigid herd inspection but in both cases I feel that the public have a right to the added safeguard of pasteurization.

Undulant fever is a disease with a very low mortality but when man is infected with it he may become very ill and experience a long and expensive convalescence. I believe this disease will eventually prove to be a boon to humanity if the fear of it will tend to bring about universal pasteurization for that would automatically end our troubles with respect to milk-borne diseases, many of which are worse than undulant fever. I feel that now is the psychological moment for public health workers to put over pasteurization of all milk.

I sometimes wonder if the tuberculin testing of cows has not set back the hand of the clock in public health in so far as people have come to look upon milk from tuberculin-tested cows as safe and not requiring the additional safeguard of pasteurization. In Indiana we often find it hard to talk pasteurization to many people. They will often buy milk produced in a dirty barn and under undesirable conditions in preference to pasteurized milk. In some of the smaller towns throughout the State, a good dairyman will produce a good clean product and sell it without pasteurization. A dairy in town will buy milk from every available source and often of questionable purity and in order to make it keep will pasteurize it. Under such conditions, the physician has a good argument to turn down the pasteurized product in favor of the raw milk but even at that I believe the pasteurized milk is the safer of the two.

You can talk about producing clean and safe milk by the method mentioned by Dr. Giltner, the supervision of the herds, etc., to large institutions such as the certified milk dairies and they will produce good and fairly safe milk but what can you do about training the average farmer who, among other interests on his farm, produces milk from half a dozen or a dozen cows? He has not the time, the money or inclination to use the necessary refinements of the dairy industry, but can nevertheless produce a reasonably clean product fit for human food but it should be safeguarded by pasteurization.

You can and should go on and clean up the herds of tuberculosis and undulant fever but while you are doing it let us safeguard the public health by pasteurization of milk from all herds. Why not pasteurize milk? You do not hesitate to cook meat and vegetables and pasteurized milk is heated to only 145° F. We are told pasteurization destroys the vitamins and it is more or less true, but milk is not a reliable source of vitamins. In the summer time, when the cows are on pasture, no doubt, milk is rich in accessory food factors but not so in winter when the cattle are stall-fed. Vitamins are of importance to children and invalids living on a restricted diet but to the average individual on a mixed diet, the loss of vitamins in the milk used is quite insignificant. In the diet of children, the lack of vitamins can be made up easily by supplementing the diet with cod-liver oil, orange juice, etc.

Until we can control the large human factor involved in the production of milk, which time will probably be long in coming, we must either safeguard our milk by pasteurization or continue to suffer from milk-borne disease.

Doctor Pearson a Perpetual Booster

Dr. Raymond A. Pearson, president of the University of Maryland and executive officer of the Maryland State Board of Agriculture, continues to say a good word for the veterinary profession whenever the opportunity is offered. At the first annual banquet of the Baltimore Livestock Exhibition Company, held in Baltimore recently, Dr. Pearson made the following statement:

> The veterinarian is the keystone in the arch which supports the live stock industry. Without the live stock industry, agriculture could not succeed and the country at large depends on agriculture.

Veterinary School at Berne, Switzerland.

RECENT PROGRESS IN OUR KNOWLEDGE OF MILK FEVER*

By PIERRE A. FISH

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In the list of animal diseases there is probably none more saturated with theories than milk fever. Aside from clinical symptoms, until recently, very little in the way of demonstrable evidence has been brought out relative to the internal changes in the organism. In 1923, Hayden and Sholl,[1] after a study of fourteen cases, concluded that a hyperglycemia existed. In 1924, having examined thirteen additional cases, Hayden[2] reaffirmed the previous conclusion. In 1925, Widmark and Carlens[3] proposed a theory of a deficiency of blood sugar or a hypoglycemia, basing their view upon experimental evidence in which cows treated with insulin apparently presented some symptoms analogous to those found in milk fever, and that intravenous injection of 3 to 5 liters of 5 per cent glucose solutions brought about apparent recovery. The udder was likewise inflated to guard against a relapse. They also demonstrated the interesting fact that udder inflation increased the amount of sugar in the blood and, since the inflation is considered responsible for the cure, it might be inferred that the increased sugar, overcoming the hypo-glycemia, was a factor in bringing about the desired result. In their published report there were included some cases in which there was a distinct hyperglycemia before treatment. This difficulty was explained on the assumption that the glucose content was low but lactose passed from the mammary glands into the circulation and thus increased the total sugar content. It was suggested that this explanation might apply to the results obtained by Hayden.

The soundness or unsoundness of the theory was dependent upon some method by which the glucose and lactose in the blood might be differentiated from each other, and at that time such a method was not available. Folin and Svedberg,[4] applying the method of fermentation, were able to throw light on this difficulty and Hayden,[5] testing this method on the blood of milk fever cases, was able to show that in the majority of the cases lactose was not present in the blood in appreciable amount before the udder

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was inflated. After inflation, a sugar not fermented by yeast (lactose) appeared in the blood in marked quantity and there was likewise an increased amount of glucose. These results were contrary to the hypoglycemic theory, since they indicated that the hyperglycemia is due in part to an increased amount of glucose, and that for a few hours after inflation there was a still further increase of glucose as well as lactose. The further increase of glucose presumably was due to defunctionizing the gland temporarily as a result of inflation, so that the glucose ordinarily used in the production of lactose remained in the blood. The work of Little and Wright, Moussu, Schlotthauer and others brings confirmatory evidence that a higher percentage of sugar is usually present in the blood of milk fever patients before treatment, and there is, therefore, an increasing tendency to reject the view that a hypoglycemia is the causative factor. Based on our own experience and the methods employed, we believe that the average range for sugar in the blood of normal cows is from 40 to 60 mg. per 100 cc. In thirty-five cases of milk fever examined in our laboratory, only three showed a sugar content below 40 mg., ten were in the normal range (40 to 60 mg.) and 22 cases were found with the blood sugar ranging from 60 to 200 mg. per 100 cc.

A *second* instance of demonstrable change of internal conditions relative to milk fever was brought out in a paper by Little and Wright,[6] in 1925. Their investigation originated in the observation that the tetany of milk fever presented very similar clinical symptoms to those found by other workers in connection with the lowered calcium content of the blood of parathyroidectomized animals. Twelve cows "down" with milk fever gave an average of 5.23 mg. of calcium per 100 cc of blood, which represents a reduction of about 50 per cent of the amount found in normal cows (9 to 11 mg. per 100 cc).

In 1925, just prior to the appearance of Little and Wright's article, Dryerre and Greig[7] advanced the theory that milk fever is due primarily to a relative parathyroid deficiency. Their theory, at that time, was independent of any experimental evidence. Although recognizing the parathyroid function of regulating the amount of calcium in the blood, they stressed the idea that a disfunction of the parathyroids resulted in a guanidine intoxication as the principal cause of milk fever.

Work in our laboratory, by Hayden,[12] indicates that the amount of guanidine in the blood of milk fever patients does not

differ materially from that found in normal cows, and justifies, in our minds, the opinion that guanidine is not an important factor in the causation of the disease.

The deficiency in blood calcium, first demonstrated by Little and Wright, has been confirmed in our laboratory. An examination of 27 cases of milk fever gave a range varying from 1.75 to 6 mg., with an average of 3.31 mg. of Ca per 100 cc, which is appreciably lower than that obtained by Little and Wright.

A later paper by Dryerre and Greig[8] gives additional confirmation to the demonstration of the deficiency in blood calcium. In an examination of 40 samples of blood of milk fever cases they found a range of 3.35 mg. to 7.76 mg. with an average of 5.18 mg. Ca per 100 cc—about the same as that obtained by Little and Wright.

A *third* instance of demonstrable evidence was noted by Fish,[9,10] who found a deficiency of the blood phosphates in milk fever patients. The intimate relationship existing between calcium and phosphates in many of the body processes suggested the idea that the phosphates might be affected as well as the calcium. In the tetany of parathyroidectomized animals there is a reduction in the blood calcium but an increase in the phosphates. In rickets there is no reduction in the calcium but there is a decrease in the phosphates. In milk fever there is a reduction in both calcium and phosphates.

In 25 cases of milk fever, Fish found an average of 2.39 mg. per 100 cc of inorganic phosphates, as against 4.65 mg. for milk cows, or 6.25 mg. for dry cows. Eighteen of the cases showed a range from 1 to 3 mg. per cent; six cases, from 3 to 4.25 mg. per cent and one case less than 1 mg. per cent. The acid soluble phosphorus tests (24 cases) showed twelve patients with a range of 5 to 7 mg. per cent; seven patients showed a higher range and five patients were below 5 mg. per cent. The average obtained from representative milk cows was 7.96 mg. per cent and for dry cows 9.32 mg. per cent. Parathyroidectomy and rickets show that a diminution of either blood calcium or phosphates does not necessarily induce a reduction of the other constituent. When both constituents are reduced it might be expected that the effects would be more severe.

Fish also noted that after inflation of the udder the phosphates returned more rapidly to their normal amount than did the calcium. Within six or eight hours, the normal standard had been reached and in that period many of the patients were upon

their feet again. While this may be only a coincidence, it is also conceivable that a normal phosphate balance in the blood may be a very important factor in effecting a cure. In a number of the cases the calcium was still below normal after 24 hours. Aside from its possible relations with the calcium in the body processes, a deficiency of the phosphates may affect the buffering quality of the blood and likewise affect the hexose-phosphate relationship in the muscles, a phenomenon concerning which much is still to be learned.

A very up-to-date and interesting paper on milk fever and associated conditions was published by B. Sjollema,[11] early in 1929. (The original paper was printed in the Dutch language, in the fall of 1928). The paper is of considerable importance because it represents a study of an extensive list of the constituents of the blood. The most marked changes were found in the calcium, phosphates and sugar. The potassium, magnesium, chlorides, cholesterin and alkali reserve of the blood did not vary materially from the normal. In several cases the creatin, creatinin, urea and acetone were increased. Sjollema seems to have been unaware of the prior work of Little and Wright, in 1925, demonstrating the reduced blood calcium in milk fever and of the theoretical observations of Dryerre and Greig relative to a disfunction of the parathyroid glands. In his paper he states that an English investigator visited his institution in September, 1928, and told several of Sjollema's colleagues that he had demonstrated a marked reduction in the calcium content of the blood serum. Sjollema and Fish, ignorant of each other's results, discovered independently the deficiency in blood phosphates and were apparently working on the subject over the same period of time, 1927 to 1929.

Sjollema's data on calcium are similar to the results obtained by the investigators previously mentioned. His range was from 2.7 mg. to 7.3 mg. with an average of 5.11 mg. per 100 cc (39 cases). For the inorganic phosphates his range was from a trace to 7.5 mg., with an average of 2.25 mg. per 100 cc, which is very close to that obtained by Fish (2.39 mg. per 100 cc). Sjollema also perfected a method for differentiating glucose from lactose in the blood and has added materially to our knowledge of other blood constituents which might be involved in milk fever but apparently are not. He believes the calcium deficiency is associated with a disturbance of the parathyroid glands. Apparently not cognizant of the work of others, it is remarkable that his

results should confirm so closely other observations on the deficiency of the calcium and phosphates in the blood of milk fever patients.

Very remarkable cures are claimed by the use of calcium chlorid solutions injected intravenously. A ten per cent solution of crystallized calcium chlorid to the extent of 300 cc to 400 cc, slowly injected, is recommended for treatment. At times, the addition of one or two milligrams of adrenalin, or atropin, or sixty grams of glucose to the solution is also recommended. Recovery occurred without other forms of treatment and in a number of cases the calcium chlorid treatment was used when inflation of the udder failed to help and in other instances when the patients were in an apparently hopeless condition. Occasionally there were relapses and a second injection was necessary to secure recovery.

Briefly summarizing our present knowledge of demonstrated and confirmed evidence brought out during the last six years, there is, in milk fever, *a hyperglycemia, a hypocalcemia, and a hypophosphatemia.*

Are the number of milk fever cases increasing? The answer will undoubtedly vary in different sections of the country. The answer will depend also upon the number of high grade animals kept in the herds. The records of the ambulatory clinic of the New York State Veterinary College, covering a period of twenty years, show that more cases of milk fever were attended during the last five years than during the whole fifteen-year period preceding. During the five-year period, 153 cases were treated, as against 146 cases for the previous fifteen-year period.

The tendency is to weed out the scrubs and "boarders" and cherish the high producers. The latter are encouraged with high rations to produce the largest available amount of milk and with this procedure the tendency toward milk fever increases. Although the blood constituents may show a normal balance, there is evidence to indicate a drain on the body tissues of their calcium and phosphate constituents, against which the mineral portion of the ration is unable to afford complete and adequate compensation. With the onset of lactation in heavy producers, the drainage may be excessive enough to upset the metabolism of the animal and milk fever results.

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Discussion

Dr. C. J. Marshall: The older practitioners will remember many disappointments and surprises in their experience with milk fever. At first it was anything but pleasant or satisfactory. There was no disease of good dairy cows more resistant to our old forms of treatment. We had many theories about the cause of the disease and nearly every form of treatment known to medical science was tried with disappointing results.

The true cause remains a mystery up to the present time. The Schmidt treatment was one of the greatest victories of veterinary science. It was hailed with unusual satisfaction by dairymen and veterinarians in all parts of the world where good dairy cows are kept, and has stood the test for over thirty years. It is doubtful whether any form of treatment will ever excel the results obtained by inflation.

The veterinarian is fortunate in having such a reliable treatment. Yet, we wonder many times about the cause of the disease and why udder inflation has proven so satisfactory.

Thirty years ago, auto-intoxication was considered the best theory to account for the cause. Physiology, pharmacology and pathology are much better understood now. We are fortunate in having such excellent veterinarians as Fish, Hayden, Little and Wright, Widmark, Folin, Sjollema, Amadon, and others, devoting so much attention to the cause of milk fever.

Dr. Fish has conclusively shown that hypoglycemia is not a constant condition and that the blood sugar is most often increased rather than decreased in milk fever. Most investigators have arrived at a similar conclusion. We did have slight hopes recently that glucose might prove a valuable form of treatment. It has been tried quite extensively and results have been disappointing. Yet, apparent benefit has been observed in certain cases. Theoretically and practically it has been eliminated as a specific form of treatment by the investigators mentioned. Fish and Sjollema, working independently, have found that hypocalcemia and hypophosphatemia are constantly present in milk fever.

This discovery has suggested a new form of treatment. It is claimed that intravenous injections of 300 to 400 cc of a 10 per cent solution of calcium chlorid have been tried on over forty cases of milk fever and that results have been practically as satisfactory as those obtained from udder inflation and that cures have been obtained by calcium chlorid in certain obstinate cases when inflation had failed.

With such encouraging results, it is my opinion that calcium chlorid is worthy of a trial in all forms of milk fever, including the usual delayed and obstinate forms.

It remains to be proven whether this form of treatment will be as effectual as ordinary inflation, or can be used to best advantage alone or in combination with inflation.

Calcium chlorid crystals are not carried in stock in many drug stores. Calcium chlorid powder is listed in catalogs of supply houses. If one plans to use it, it should be weighed and kept in well-stoppered bottles. The average dose is 30 grams of calcium chlorid in 300 cc of sterile water, given intraven-

ously. The dose can be repeated. It can be given with adrenalin chlorid, 1 to 2 milligrams, and glucose, 2 ounces, if desired.

The metabolism of calcium has not yet been carefully worked out. It may be found later that a larger or smaller dose will be more satisfactory or that some other preparation of calcium may give better results.

If calcium or phosphates can be used in some way to correct the deficiency of these products in the blood, the treatment would have a much more scientific value than udder inflation, as it has been used in the past.

Dr. Fish suggests the possibility of milk fever cases increasing in number. The ambulatory clinic at Cornell has treated 153 cases in the past five years. The ambulatory clinic at Pennsylvania is seldom called to treat milk fever. These cases are usually treated by the local veterinarian. In the good milking herds with which we are familiar, milk fever is of rare occurrence. We were of the opinion that with increased milk-production a better system of care and feeding had developed and that in well-conducted dairies milk fever was being encouragingly prevented. It is now quite well known that good cows should not be milked clean soon after calving. Cows that are milked regularly up to calving time, and those that develop mastitis, seldom, if ever, develop milk fever.

We have learned to use the services of the bacteriologist when dealing with communicable diseases. We can also receive much help from the physiologist, the pharmacologist and the bacteriologist when dealing with many non-infectious diseases and especially parturient paresis. If the practitioner would avail himself of the opportunity of working with those who are qualified and equipped to do laboratory work, the benefits would be mutual. Blood analysis in milk fever is important.

I wish to congratulate Dr. Fish and his co-workers on the excellent work they have done and are doing and also on the clear and instructive report he has just given you. (Applause)

Be on Your Guard

Drs. Hufnall and Keigan, 1612 East Alabama St., Houston, Texas, desire to warn other veterinarians to be on the lookout for an impostor answering to the following description:

> Briefly, this party answers to the name of Cline or Klein, undersized and stout in stature, gives facial appearance of being Jewish, strong German accent, flat-footed, about 55 years of age, gray hair, bald in front, smooth faced, when seen last wore high black shoes, blue serge pants; greenish-hue, pinch-back coat of gaberdine material; wears neat collar-attached shirts, black four-in-hand tie, and a pearl-colored soft hat, very soiled, with ribbon heavily stained with sweat marks. This party gains the confidence and fellowship of veterinarians by his unique and rare knowledge of veterinarians in every state in the Union, their business methods, characteristics, life histories and so on. He has worked for many hospitals and, from the trend of his conversation, mostly in the East. Poses as a kennelman and professional groomer of dogs and is quite a devotee of dog shows. His conversation reveals that he has followed dog shows extensively.

If this party should show up at any veterinarian's office, keep an eye on him and wire Drs. Hufnall and Keigan, at their expense. They were relieved of a large sum of currency by this "wily and velvet-handed artist" on the night of November 1, 1929.

NOTE ON SOME URINE AND BLOOD CHEMISTRY IN MILK FEVER

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Interest in milk fever has recently been stimulated by biochemical theories and facts which have been put forward during the last five years. The extremely rapid collapse, together with the equally rapid recovery, place the disease almost in a category by itself, and make it of interest to the physiologist and biochemist, as well as to the veterinary clinician. In reviewing the findings in this condition the writer has been struck by the number of facts which seem to group themselves about a common origin:

1. The urine often contains albumin and casts; sometimes leucocytes and occasionally red blood-cells.
2. The urine often contains glucose (apart from lactose).
3. The urine is acid, whereas normally cow's urine is alkaline.
4. The blood sugar is variable, but is more often raised than at a normal low level, though sometimes a hypoglycemia is found.
5. Blood chlorides are slightly low, normal on recovery.
6. There is a raised urea and N.P.N. in the blood, both the values becoming normal on recovery.

Almost all these statements have recently been the subject of experimental inquiry at the New York State Veterinary College, and, with the exception of the finding of hypoglycemia, have received verification in those laboratories. The conjunction of such a series of findings, were they to be reported in a human pathological condition, would be interpreted as the result of a lowered blood volume, with resulting partial asphyxia. A lowered blood volume is the result of loss of fluid, either through some external agency or some internal disturbance resulting in escape of plasma through the capillary walls. In either case prompt restoration of fluid is demanded. The use of large quantities of normal saline, given either intravenously or interstitially, and the former for preference, is very extensively followed in human practice. Duodenal obstruction, intestinal paresis, vomiting of pregnancy, infantile diarrhea, are conditions in which the

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urine and blood findings are similar to those just described in milk fever and in which the administration of large amounts of fluid is now routinely followed. This condition is often called "anhydremia" or, in a looser way, "dehydration." It can pass readily in shock with its attendant symptoms of low temperature, low blood pressure, and weak rapid pulse. The amount of saline given to human adult cases of dehydration varies from 1 liter to 3 liters, though as much as 7 or 8 liters may be given in exceptional cases. This represents from ¼ to ¾ the average blood volume of the patient. Often, instead of normal saline, a 5 or 10 per cent glucose in saline is given. The glucose may be given, not on account of any inherent disturbance of carbohydrate metabolism, nor because of any blood-sugar findings, but because human patients in such a condition are often in need of immediately available nourishment, which cannot be given by mouth. Glucose by vein, when given with large amounts of fluid, is readily utilized. This is mentioned because recently there has been recommended for milk fever the use of glucose. The results appear to be far from uniformly favorable, though Widmark and Carlens obtained successful results on their original observations.

If the blood and urine findings in milk fever, given at the beginning of this paper, bear the same interpretation as they would in human medicine, they demand the administration of large amounts of fluid. In comparison they demand, as the cow's blood volume is estimated at 40 to 50 liters, an amount of saline varying from 10 to 30 liters. This may seem an enormous amount, but it certainly seems necessary vastly to increase the amount of intravenous fluid over the often recommended 200 cc of 10 per cent glucose, if intravenous therapy in milk fever is to be successful or is to receive an adequate trial. Fish has pointed out that Widmark and Carlens used 3 to 5 liters in their original therapy and offers the surmise that the bulk of fluid rather than the glucose might be the decisive factor in success. Analogy with human medicine certainly offers support to the conjecture.

In addition there have appeared other statements, which, either being overlooked, or not so susceptible to laboratory control, have not the precise value of the first series but which nevertheless point to dehydration:

1. Law states that the blackness and thickness of the blood in milk fever is well known to all observers.

2. Williams is emphatic that little or no urine is passed during the attack.

3. Menig has made observations that the urine volume, previous to the attack, is diminished and is of high specific gravity.

4. Milk flow is diminished or ceases during the attack.

These statements require further precise observation, but, if true, they bear the same interpretation as the previous series. Loss of fluid from the blood could well be the origin of all.

Finally it may be suggested that the prevalent method of cure by means of udder inflation, by at once checking any further milk-production, and by raising the external pressure and increasing the vascular tone in the udder, inhibits the further development of shock symptoms and allows time for the reserves of water present in the body to come into play and exert their natural compensatory effect.

Turkeys to Be Graded

Preparations for the government grading of more than 500,000 turkeys this year are being completed by the Bureau of Agricultural Economics of the U. S. Department of Agriculture. Government inspectors will grade birds at terminal markets in Boston, New York, Philadelphia, Baltimore, Washington, Pittsburgh, Detroit and Chicago, and cooperative agreements have been made with state departments of agriculture for federal-state grading at shipping points in Colorado, Utah, Nevada, Idaho, Wyoming, California, Montana, Minnesota, Virginia and Maryland.

Government-graded birds will have paper "bracelets" around the shank, on which the grade will be printed over the initials of the Bureau of Agricultural Economics, U. S. Department of Agriculture. The grades are "U. S. Prime" and "U. S. Choice," as applied to young birds that meet the requirements of these grades, and the older birds which qualify will be labelled "U. S. Prime, Mature," or "U. S. Choice, Mature." More than 100 classers at shipping points have been licensed by the Bureau of Agricultural Economics for grading turkeys.

Approximately 200,000 turkeys were government-graded last year, but it is expected that many more will be graded this year in view of the estimated increase of 9 per cent in the size of the crop as compared with last year.

STUDIES IN INFECTIOUS ENTERITIS OF SWINE
IV. Intestinal Coccidiosis

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INTRODUCTION

During the course of a series of studies in infectious enteritis of swine, our attention was directed from time to time to cases characterized by a low-grade intestinal inflammation and erosions that were clinically designated as enteritis. On the basis of pathological observations, they could not be placed in the same class with the characteristic infectious enteritis cases caused by the *S. suipestifer* and the secondary invader, *Act. necrophorus*, usually referred to as necrotic enteritis. That these atypical or questionable cases might be chronic, low-grade forms of suipestifer infection did not appear probable, because of the negative serologic and bacteriologic findings. A diagnosis of enteritis on the basis of clinical findings appears correct only if the term enteritis is used in a collective sense and not to indicate a specific infection.

In addition, the frequent histories of unthriftiness in pigs before and after hog cholera vaccination cannot be explained from an etiological standpoint. Infection with the suipestifer organism or ascaris infestation is not an answer to all such swine troubles. When the question, "Can vaccination troubles be caused by suipestifer infection?" was put to us, we were inclined to answer that every case must be judged on its own evidence. We are now convinced that this is true. History shows that when the specific causal factor and an accurate knowledge of a disease process are not known, confusion and errors result. The erroneous impressions held before the demonstration of hog cholera virus afford an illustration. Without knowledge of specific etiological factors, several or more diseases may be attributed to one cause. Further illustration of this is furnished in the case of Johann Georg Roederer[3] and his assistant, Wagler, in their study of typhoid fever during the 1757-63 outbreak in Göttingen. They made careful autopsies and studied the lesions carefully from a gross standpoint, the only method then available, and concluded that typhoid was identical with intermittent fever

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and dysentery. Our conception of typhoid was not placed upon a scientific basis until after the causal organism was noted by Eberth, in 1880, in the tissues of typhoid fever victims, and after Gaffky, in 1884, furnished definite proof of *B. typhosus* as the etiological factor.

The uncertainties concerning the types of infection in swine mentioned above suggested a broad field of investigation and with this in mind a number of herds, where both diagnosed and undiagnosed swine troubles had occurred, were included in a survey study. These herds we planned to visit periodically or as occasion arose. This was made possible through the cooperation of Doctors F. E. Walsh and W. F. Chivers, of the Ambulatory Clinic, who brought us in touch with such herds and gave us the benefit of their clinical observations and knowledge of the histories of such herds in previous years. This cooperation plays an indispensible part in the conduct of this project and we desire to emphasize the importance of clinical and laboratory cooperation. Our studies in accordance with such a survey plan were confined to only twelve premises in the beginning, to enable systematic detailed observations. As a preliminary step, unthrifty or sick pigs, when available, were brought to the laboratory and subjected to bacteriologic, serologic, parasitologic and pathologic studies. In addition to recording histories and notes regarding location of buildings, yards, etc., composite and individual fecal specimens and soil samples were examined for parasite ova. This examination was made by the centrifuged sugar-flotation method originated by A. L. Sheather, of London, with slight modifications as described by Doctor E. A. Benbrook.[24]

During the course of these survey studies, we were surprised to find, in addition to various parasite ova, the almost constant presence of coccidia in the soil and in the feces of swine from the herds under observation.

The following report is a résumé of our field studies, together with findings in experimentally induced cases of swine coccidiosis. In order to record, for comparative purposes and study, the number of oocysts found in our material, the preparations were made in the usual manner as described by Benbrook[24] and the approximate number of oocysts present in the low-power field were noted. When the coccidia were present in small numbers, counts were made direct from low-power fields, but when in large numbers the average of about 20 high-power fields was

determined and multipled by the factor 16, giving approximately the number present in the low-power field.

Literature

A brief summary of the literature dealing with coccidiosis of swine is essential as a basis for further studies on this apparently important but heretofore overlooked disease of swine in America. The literature dealing with coccidiosis in swine prior to 1920 is of interest, notwithstanding the inadequate descriptions and confusion prevailing as to classification. Zürn,[1] in 1878, and Johne,[2] in 1882, report having found coccidia in the intestines and liver, respectively, of swine. In 1896, Wasielewski[3] designated the coccidia of swine and calves as *Eimeria zurni*. Voirin,[4] in 1900, reviewed the findings of Zürn and Johne, and took up the morphology and biology of some forms of coccidia. According to Nieschulz,[18] Neveu-Lemaire, in 1912, refers to the swine coccidium as *Eimeria jalina*. *Eimeria jalina* traces back to a report by Perroncito, in 1902. Perroncito's publication was not available to us but later authors, Brumpt and Nieschulz, state that Perroncito dealt with a parasite encountered in man, swine and guinea pigs, which they believed to be a blastocystis and not a coccidium. The texts by Ostertag,[6] in 1907, and Kitt,[7] in 1908 and 1910,[8] speak of Johne's report of coccidiosis in the liver of swine but make no mention of the intestinal form. Schultz,[10] in 1918, states that all classes of animals may be attacked by this type of protozoa (coccidia) which often cause diseases among rabbits, mice, moles, sheep, goats, cattle, swine, etc. His paper deals with losses among cattle in the Pacific Northwest (United States) and does not specifically describe findings of coccidia in swine.

Douwes,[11] in November, 1920, reports having observed coccidia in swine a number of times. He claimed to be able to distinguish those of swine from the *Eimeria stiedae*. Douwes believes two species occur in swine, a large species measuring about $50\mu x33\mu$ and a small species. He does not name either of the supposedly two species from swine. On January 19, 1921, Douwes' Inaugural Dissertation[12] appeared, reporting the occurrence of coccidia in pigs from 3 to 10 weeks of age. In other subjects, 12 weeks old, he found coccidia present in very small numbers and sometimes entirely absent. He did not find them in fully grown swine. Their pathogenicity is questioned by Douwes because the pigs studied by him did not appear to show any deviation in health,

but he qualifies this statement by adding that in coccidial infections, cells are continually destroyed and in that sense coccidia are pathogenic. As in his previous report, he believes that a large and a small species occur. Douwes finds the small variety (17-22μ long and 14-19μ wide) is of widespread occurrence in swine. This form he calls *Eimeria debliecki*. In a very small number of cases he believed an infection to be due to a form having oocysts 50μx35μ. The so-called large variety is not named by him. Douwes' forms incubated in the moist chamber proved to be Eimeria.

In February, 1921, Nöller[13] found a considerable number of coccidia in one of 60 swine slaughtered at the Hamburg abattoir. They were mostly 10-12μ wide and 15-20μ long, with some as long as 33μ. These he named *Eimeria suis* and claimed priority over Douwes' published report (January, 1921)[12] on the basis of an oral presentation in a scientific gathering, December 7, 1920. Krediet,[14] on January 28, 1921, at Leiden, found 11 of 50 pigs eliminating coccidia in the feces. He measured a large number of oocysts, the smallest being 12μx12μ, the largest 18μx35μ. Krediet used the name *Eimeria jalina*, not having seen Douwes' inaugural dissertation of January 19, 1921. Nöller and Otten,[15] in 1921, by means of the concentrated sodium chlorid method, found 15 out of 20 mature swine at the Berlin abattoir positive to coccidia, and conclude that mature swine can be carriers, resulting in the spread of infection among young swine. Cauchemez,[16] in December, 1921, found swine infected with coccidia, 13-30μ long and 9-18μ wide, at the Vaugirard abattoir and uses the provisional designation *Eimeria brumpti*. The proportion infected was noticeably greater during the summer, decreasing during the autumn. Because the mature swine apparently remained in excellent general condition, these sporozoa seem to be regarded as harmless by Cauchemez. His experimental feeding of sporulated oocysts resulted in the elimination of non-sporulated forms in the same manner as in our own feeding experiments described in this paper, except that in our cases an overwhelming number of oocysts were sometimes discharged and the pigs became emaciated, presenting characteristic clinical and postmortem deviations. The condition of the culture or the number of sporulated oocysts in Cauchemez's cultures might explain the mild symptoms. We have found that the condition of the culture and the age of the pig fed are important. Our experiment pigs were

older than those used by Cauchemez. Cauchemez adds in his report:

> Perhaps other observers forewarned will be able to refer to coccidiosis in very young pigs as some serious intestinal manifestations which we have not discovered in adult swine killed at the Vaugirard abattoir.

Nöller and Frenz,[17] in 1922, encountered two pigs, aged 10 days, infected with coccidia. No pathogenic bacteria nor evidence of a filtrable virus were revealed. A hemorrhagic inflammation of the intestines, especially marked in the jejunum of one, characterized these pigs. Both oocysts and merozoites were found. The authors believe that previous reports of great losses observed in this herd probably trace back to this infection by coccidia. As in Nöller's first publication, they adhere to the designation *Eimeria suis* and state that on the basis of size their findings correspond to the small coccidian forms of Douwes (*E. debliecki.*) Nöller and Frenz attribute death to this coccidian infection and point out that death can intervene even before large numbers of oocysts are formed. Nieschulz,[18] in 1922, takes up the question of naming the coccidia from swine. He concludes that *Eimeria debliecki* (J. B. Douwes, January 19, 1921) has prior claim over all other designations. Cauchemez,[19] in 1922, retracted the designation *Eimeria brumpti* provisionally used by him in 1921. Subsequent to Cauchemez's first paper,[16] in 1921, there came to his attention the publication of Douwes,[12] using the designation *E. debliecki*, and that of Neischulz,[18] justifying that designation, after which he concludes:

> The name *Eimeria debliecki* must then alone persist, *Eimeria suis* (Nöller) and *Eimeria brumpti* (Cauchemez) becoming synonyms.

Sheather,[20] in 1925, in England, reports the presence of coccidia in the intestines of two young pigs. He measured 100 oocysts from the first pig. They ranged from 13μx16μ to 23μx30μ. After placing them into four arbitrary groups according to size, he states:

> It would appear to indicate that, if size is sufficient to distinguish species, two different coccidia were present.

Munnik,[21] in 1924, in the Netherlands, found 58 of 100 samples of swine feces from March, 1921, to March, 1922, positive for coccidia. The oocysts varied in length from 14μ to 35μ and in width from 11μ to 29μ. The mean length and breadth were 24μ and 18μ, respectively. Instead of grouping them according to length and width, Munnik plotted them, based on volume expressed in cubic microns. About 60 per cent were of the

middle-sized forms. Between the largest and smallest there were intermediate sizes and nothing in the plotting suggests more than one variety. Davis and Reich,[22] in 1924, report the presence of oocysts in the intestinal contents of five slaughtered swine from West Oakland, California. After some unfruitful morphological comparisons with the oocysts of *Eimeria zürni*, they state in part:

> After the study of oocysts in the cases of swine coccidiosis we are left in considerable doubt as to their significance. There are some points of difference between this form and that found in the cattle examined, but there is an overlapping borderland of similar forms. The scavenger habits of the hog make it peculiarly liable to chance infections; it is quite possible that swine pastured with cattle harboring *Eimeria zürni* might acquire an infection.

On the basis of these observations and without experimental feeding they dispose of this problem in swine by the following comment:

> That coccidiosis of swine is not of pathogenic nor of economic importance is suggested by the fact that veterinarians are not cognizant of its existence.

They were not able to find any literature on coccidia in swine, but in an addendum, after their paper had gone to press, they note Sheather's paper containing a report on swine coccidiosis.

De Graaf,[23] in 1925, reported finding 257 of 500 samples of swine feces from November 22, 1923, to March 6, 1924, positive for coccidia. He described his forms under the name of *Eimeria deblieckí* and gives detailed cuts showing morphology and sporulation. Yakimoff et al, [27,28] in 1926 and 1927, report on the occurrence of coccidia in swine in various sections of Russia. In some groups of swine as many as 45.4 per cent were infected. Sporulation tests showed their forms to be Eimeria and they refer to them as *Eimeria deblieckí* (Douwes, 1921). They report constipation and desquamation of cells in one group. As a result of their findings up to 1927, they believe without question that coccidiosis is widely distributed throughout Russia. In addition to the literature mentioned, the text-books by Wenyon,[26] Hutyra and Marek,[25] and Glässer[29] make reference to one or more forms of swine coccidiosis.

Prior to the appearance of Douwes' paper, on January 19, 1921, there are not sufficient data on which to establish a designation. The name *Eimeria jalina* used by Neveu-Lemaire is eliminated because it is based on Perroncito's work in which a blastocystis was mistaken for a coccidium. Douwes' first paper on swine coccidiosis in 1920 does not use a designation, so establishes no name, but his inaugural dissertation, as of January 19, 1921,

using the name *Eimeria debliecki* for his small form, together with an adequate description, establishes priority. Nöller's paper was not published until February, 1921, but he mentioned his findings in an open meeting in December, 1920, using the name *Eimeria suis*. According to the rules of priority, the published date of a paper and not the oral presentation establishes the claim. *Eimeria jalina*, reserved by Krediet, is ruled out, since he was unaware of Perroncito's work wherein he apparently dealt with a blastocystis instead of a coccidium. In 1921, Cauchemez proposed *Eimeria brumpti* for the coccidium of swine, not having seen the work of Douwes or Nöller. In 1922, after having had access to these papers, he withdrew the designation *Eimeria brumpti* and recognized *Eimeria debliecki*. Yakimoff arrived at the same conclusions as Nieschulz, on the prior claim of *Eimeria debliecki* (Douwes).

The coccidia found in the swine of Iowa, we believe, are identical with *Eimeria debliecki* and we shall provisionally use that designation on the basis of our observations which follow.

Field Studies

Farm A—Story County, Iowa: Pigs had access to all fields and yards on the farm. Drainage and sanitary conditions were very poor. The pigs were farrowed from the last day of February until the second week in April. On July 6, 1928, the spring pigs were in poor condition. Most of them were emaciated, weighing from 20 pounds up, and some were scouring at the time.

Specimen I: A composite fecal sample from these pigs showed about 30 oocysts in each low-power field made from a sugar-flotation preparation. Ascaris eggs also were present in a similar number.

Specimen II: Mud taken from a wallow also contained about 15 oocysts per low-power field, some of which were in the sporoblast stage.

Specimen III: Composite samples of sow feces also contained between 10 and 15 oocysts.

On July 10, 1928, three typically affected pigs were brought to the laboratory for study. Fecal examination revealed the following results:

Pig 1—Few oocysts and ascaris ova

Pig 2—10 oocysts per low-power field

Pig 3—30 or more oocysts per low-power field

Pig 3 was an emaciated subject but showed no outstanding changes to account for its physical condition.

Flotation preparations made from various portions of the gastro-intestinal tract showed no oocysts in the stomach contents, none in the duodenum, a few in the jejunum, with increasing numbers in the ileum, cecum and large colon, the rectum containing about the same number. Definite counts were made in pigs described later bearing out these observations. Material for histologic study was taken from the duodenum, jejunum, ileum, cecum and large colon. Stained by the routine hematoxylin and eosin method, intracellular invasion could not be ascertained.

Pigs 1 and 2 were placed on clean ground and moved three times. The number of oocysts passed in the feces continued to decrease upon every examination. On July 16, 1928, the blood of pigs 2 and 3 was examined for suipestifer agglutinins with negative results.

Specimen I: Taken after a very heavy rain, July 18, 1928, from the same wallow as sample II of July 6, contained only isolated oocysts. The surface mud had been washed off, leaving a dense mass of fibers and corn-stalk residue.

Specimen II: Dark green stagnant liquid taken from a concrete wallow contained 3 oocysts in one preparation and 2 in a second.

Specimen III: Composite sample from pigs—30 or more oocysts per field.

Specimen IV: Composite sample of horse feces was negative for coccidia. Only several mange mite ova were found.

Specimen V: Composite sample of chicken feces taken from dropping-boards showed only one large form containing two sporoblast-like bodies.

Specimen VI: From a pig designated as No. 4, brought to our laboratory, showed 35 to 40 oocysts per low-power field. The length of the oocysts from the case ranged from 16μ to 24μ, the width from 12μ to 16μ, with an average length of 20μ and average width of 15μ. This pig was not expected to live much longer. It was placed in a cement-floored pen which was washed every few days. Cultures of coccidia were made from the feces of this subject but after about one week the number of oocysts passed became so small that it was discontinued as a source of further cultures. On August 8, the blood was negative for suipestifer agglutinins. On October 22, 1928, this subject was

released from this project, the feces being negative for oocysts. The pig was still living on January 22, 1929, stunted and with a long haircoat.

Specimen VII: Composite hog feces showed about 35 oocysts per low-power field. August 8, 1928, pig 5, brought to the laboratory from this farm the same time as pig 4, showed no suipestifer agglutinins. This subject, like pig 4, passed oocysts, was in poor condition and not expected to live. During the latter part of August it began to improve. Like pig 4, the number of oocysts eliminated in the feces decreased until finally none could be demonstrated. During the months of August and October, a number of composite and individual fecal samples examined from this farm confirmed the above-described findings. No fall pigs were farrowed this year, which probably accounts for the small number of oocysts found during October and November.

On November 6, 1928, a soil sample from wallow showed only one oocyst in every 6 or 8 fields. These possessed granular centers with some containing sporocysts. Other individual and composite samples of feces contained from 5 to 10 oocysts in each low-power field. A few ascaris and esophagostomum ova also were present. Cultures made by various methods proved these oocysts to be Eimeria, possessing four sporocysts, each having two sporozoites.

Farm B—Story County, Iowa: Our attention was called to this herd by Doctors Walsh and Chivers. A number of the spring pigs were sick. Four died or were destroyed. From the description given us, a "low grade" enteritis was inferred. On August 20, 1928, a pig weighing about 35 pounds was received from this herd. The large intestine especially was the seat of an inflammation and exudate but a true caseated membrane was absent. The wall, however, was greatly thickened. Flotation preparations were made from contents of the different portions of the intestine. A considerable number of oocysts were found. Samples from the large intestine showed from 15 to 20 oocysts per low-power field. They presented considerable variations in size (figs. 1 and 4). Unfortunately this carcass had been collected by the disposal truck before the above-described flotation preparations were completed and examined. The intestinal material which should have been subjected to histologic study, in view of the gross character of the wall and the presence of a considerable number of oocysts in the feces, thus was lost.

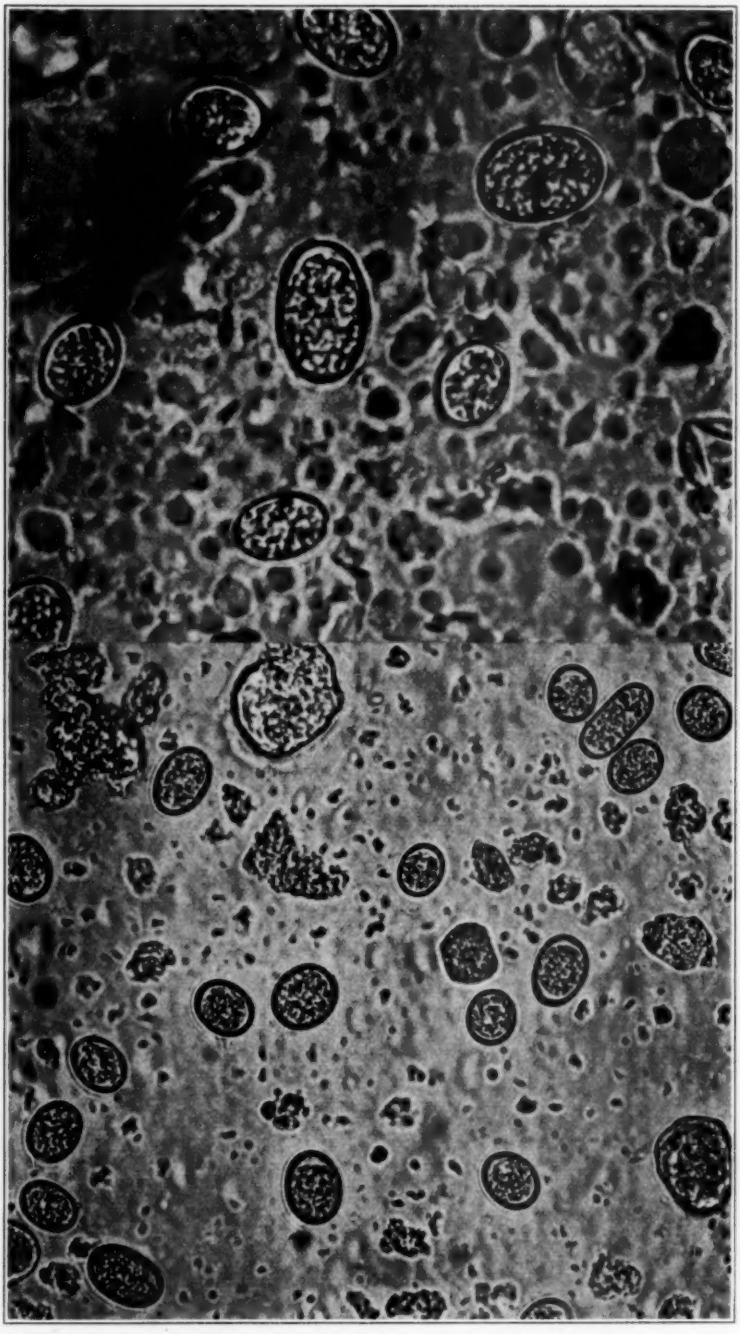

FIG. 1. (Above) Oocysts in swine feces from farm B, showing variation in size and details in newly formed oocysts. x 800.

FIG. 2. (Below) Oocysts from pigs from farm I, showing some forms encountered with variations in size. x 400.

On August 25, 1928, this farm was visited and described in detail. In brief, the drainage was very poor and sanitary conditions were likewise poor. During heavy rains some of the pastures and parts of the yards were under water. For at least nine years all classes of live stock had been given free range of the premises, without any attempt at rotation of lots. Suggestions regarding sanitation and management did not evoke much, if any, interest on the part of the owner. During the course of the first visit he stated in a pessimistic vein that each year more difficulty was experienced in raising hogs. Prior to this visit, some of the typically affected pigs had been disposed of and the balance placed on medicinal treatment by the Ambulatory Clinic staff. Only several runts which were not typical of the previous trouble were available for study. One of these subjects was brought to the laboratory. Upon autopsy it was found to harbor a number of ascaris, 10 to 12 inches long. A chronic catarrhal enteritis prevailed. Flotation preparations of the gastro-intestinal contents gave the following results:

Stomach—no oocysts
Duodenum—none
Jejunum—2 to 3 per low-power field
Ileum—about 5 per low-power field
Cecum—about 5 per low-power field
Large colon—about 10 per low-power field

Ascaris ova were present in large numbers in all preparations, the stomach containing only a few isolated ova. Mud and stagnant water around the drinking-fountain showed several oocysts per low-power field. One live and one dead chicken also were obtained. The dead bird showed advanced fowl cholera, with outstanding lesions of localization in the liver. Autopsy of the live bird revealed generalized tuberculosis. Both birds showed 15 or more oocysts per low-power field by means of the sugar-flotation method. A composite fecal sample from the cattle on these premises contained 1 to 2 oocysts per low-power field, somewhat larger than those from the swine. The average width and length were 22μ and 30μ respectively. A composite fecal sample collected from different parts of the barn-yard contained 5 or more oocysts per low-power field.

On October 9, 1928, we again observed this herd. Since our last visit, the owner had placed his spring pigs in a sweet clover pasture. These pigs had made such a remarkable improvement that we did not recognize them as being the same herd observed

several months previous. The owner seemed greatly pleased over the results obtained by moving them to clean ground and stated that he had been skeptical of our suggestions but was now convinced of the importance of lot rotation. One spring pig which had not made gains like the others, although in fair condition, was brought to the laboratory at this time. Fecal examinations of this pig were as follows: Coccidia were present but not in large numbers. They were difficult to demonstrate because of a thick fat-like film coming to the surface in the flotation preparation. On October 10, 1928, this pig showed about one oocyst in every 3 or 4 fields. Possibly many more were present but visibility was affected by a heavy film on the surface. Some oocysts showed a large clear area around the granular central mass. Fecal examinations from this subject were made every second day until October 29. The number found diminished and in some of the later examinations no oocysts were demonstrated.

During the visit of October 9, 1928, the fall pigs farrowed September 10, 1928, were in a corn-field although having access to the old lots near the buildings. The pigs were in apparently good condition. Composite fecal samples from these fall pigs showed oocysts in considerable number but owing to the heavy surface film in the flotation preparations counts were not made. Slides made from a second, third and fourth harvest were more free from the fat-like film containing flocculent material. Oocysts in these preparations were measured. In one such sample the following measurements were recorded:

Minimum	*Maximum*	*Average*
Width 14μ	Width 20μ	Width 17μ
Length 18μ	Length 25μ	Length 21μ

Another preparation from the same source revealed the following measurements:

Minimum	*Maximum*	*Average*
Width 14μ	Width 20μ	Width 17μ
Length 20μ	Length 28μ	Length 24μ

On October 16, 1928 further observations were made. Quite a number of the fall pigs were found in the lots around the buildings where the spring pigs had been during the summer. A number of these small pigs were scouring. Different samples of fresh feces collected from such pigs showed from 2 to 10 oocysts in each low-power field, notwithstanding the fatty film. Undoubtedly more were present but were rendered invisible. Owing to

the fatty film it was necessary to press down the cover-glass to render the field transparent, reducing the reading which would have been obtained by the usual technic. Cultures made from this material proved these oocysts to be Eimeria.

Farm C—Boone County, Iowa: This herd was first studied on June 22, 1928, in connection with a survey of enteritis in swine. The pigs were not in good condition and the attending veterinarian held up vaccination for hog cholera until improvement should be apparent. Fecal samples from this herd showed from 10 to 12 oocysts in each low-power field. Measurements were as follows:

Minimum	*Maximum*	*Average*
Width 14μ	Width 28μ	Width 19μ
Length 17μ	Length 32μ	Length 24μ

Cultures made in 1 per cent potassium dichromate demonstrated these to be Eimeria.

Farm D—Story County, Iowa: This farm has a long-time history of unthrifty pigs and acute enteritis. Since all of the losses could not be attributed to suipestifer infection, and in view of our findings in other herds, it was decided to make an examination for the presence of coccidia.

On October 15, 1928, composite fecal samples from older hogs showed 8 oocysts in each low-power field, and soil sample from wallow, 3 to 7 oocysts of various sizes and stages of development in each low-power field. Fall pigs in barn showed one oocyst in every 2 or 3 fields.

On November 6, 1928, some of the fall pigs were scouring.

Specimen I: Feces from one pen of these suckling pigs showed over 1000 oocysts in each low-power field. This determination was made by counting about 20 high-power fields and multiplying by the factor 16. This preparation was very dilute, more water having been added to this fecal sample than others in making the emulsion. Otherwise a still higher count would have been obtained.

Specimen II: Feces from several other pens of suckling pigs showed 10 oocysts per low-power field.

Specimen III: Composite fecal sample from mature hogs and spring pigs—about 2 oocysts per low-power field.

On November 8, 1928, two suckling pigs, about 8 weeks old, were obtained for autopsy. Both were stunted. About 7 oocysts per low-power field were found in the feces of the first

pig. Only a few in each low-power field were present in the second pig. The *S. suipestifer* could not be recovered from the spleen and mesenteric lymph-nodes of either pig. The blood was negative for suipestifer agglutinins. No gross lesions of any significance were found. On November 9 and 23, 13 samples composed of soil and feces from different groups of pigs were examined. All contained oocysts up to as high as 5 per low-power field. In the soil sample some sporulated forms were present. In a preparation from suckling pigs on November 23, 1928, one form containing four sporoblasts was noted. On November 23, 1928, a spring pig in poor condition with diarrhea and cough became available for autopsy. An extensive catarrhal pneumonia with lung worms was found. The only relevant change in connection with the present problem was a catarrhal inflammation of the cecum and large colon with some adhesions of the contents. The bile and duodenal contents were negative for coccidia.

Ileum—about one oocyst in every five fields
Cecum—2 to 4 oocysts in each low-power field
Large colon—2 to 3 oocysts in each low-power field
Rectal contents—about 8 oocysts in each low-power field.

Farm E—Boone County, Iowa: This herd has a history of infectious enteritis several years previous. During June and July, Doctor Walsh, of the Ambulatory Clinic, directed the management of these hogs with a view to getting them in proper condition for hog cholera vaccination. On the date of our first visit, our attention had not yet been directed to the possible significance of coccidiosis in swine. Our next contact with this herd was on October 3, 1928. In the meantime the herd had improved remarkably. Fecal samples taken at random from the lots adjoining barns showed only one oocyst in every 2 or 3 low-power fields. The following measurements were obtained from this sample.

Minimum	*Maximum*	*Average*
Width 14μ	Width 15μ	Width 14μ
Length 15μ	Length 20μ	Length 18μ

Farm F—Story County, Iowa: This herd carries a history of post-vaccination trouble and some unthrifty pigs during the preceding few years.

On October 3, 1928, a composite fecal sample from some of the boars in this herd showed about one oocyst in every 7 or 8 low-

power fields. The following measurements of the oocysts were obtained from this preparation:

Minimum	*Maximum*	*Average*
Width 14μ	Width 20μ	Width 18μ
Length 15μ	Length 28μ	Length 24μ

On October 15, 1928, some of the fall pigs were scouring. Eight to 10 oocysts per low-power field were present.

On November 6, 1928, a few of the fall pigs were not so thrifty as the rest. On this date, 3 to 4 oocysts per low-power field were found in the feces.

Farm G—Boone County, Iowa: This herd was included in our survey on enteritis because of previous history of disease. Our attention was first directed to coccidiosis in this herd on October 24, 1928. Two sick spring-farrowed pigs were encountered. One of these was available for autopsy. The animal was down and trembled. Respirations were increased. No diarrhea. Temperature, 100.4° F. Autopsy revealed a catarrhal pneumonia involving nearly a third of one lung. The contents of the cecum were semisolid and areas of hyperemia were present on the mucosa. The contents were very adherent to the mucous membrane, especially in the region of the ileocecal valve. The large colon presented a similar picture but in addition a great number of minute esophagostomum nodules were found. A large number of *Esophagostomum edentatum* also were found on the mucosa. Trichuris were also present as well as a large number of ascaris of different sizes. Sugar-flotation preparations of the intestinal contents were as follows:

Small intestine—few isolated oocysts
Large colon—30 oocysts per low-power field

The oocysts showed marked variations in size.

Farm H—Boone County, Iowa: This herd came to our attention some time after a history of losses following vaccination. The land was poorly drained, water standing in parts of some of the lots, several of which had the appearance of permanent wallows. Only one detailed inspection and examination of soil and feces was possible.

Specimen I: A sample of soil from one wallow at the far end of a small pasture contained a few oocysts.

Specimen II: Composite fecal sample from the mature hogs and spring pigs revealed 2 to 3 oocysts in each low-power field,

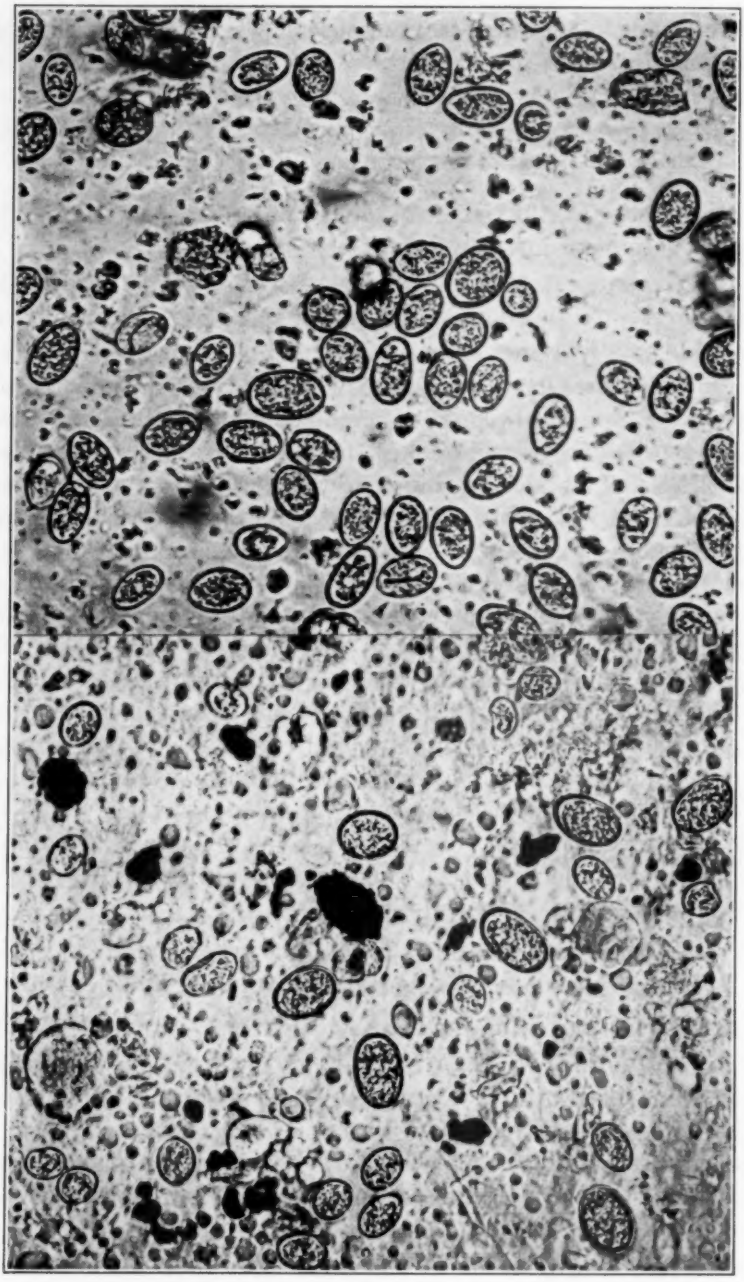

FIG. 3. (Above) Oocysts in feces of pig 3215, showing variations in size. Two very small forms present. x 400.

FIG. 4. (Below) Oocysts in the feces of pig from farm B, showing variations in size. x 400.

notwithstanding a heavy surface film on the centrifuged preparation.

Specimen III: Composite fecal sample from suckling fall pigs showed about one oocyst in every low-power field. Based upon our experience with such samples it can safely be said that a higher reading would have been obtained had the preparation been clear and undue pressure upon the cover-glass had not been necessary to render it transparent.

Farm I—Story County, Iowa: This herd consisted of spring pigs which showed outstanding variations in size. Sanitary conditions were extremely bad. The farm also included considerable low land in a creek valley. During the early part of January, an anthelmintic had been given. Subsequently the owner claimed that the anthelmintic treatment had not been effective. On February 6, 1929, treatment was repeated although no clinical indications of roundworm infestation appeared. The haircoats were slightly rough. None of the pigs was emaciated but great variations in size were noted. On this visit individual and composite fecal samples were taken. A negligible number of ascaris and esophagostomum ova were found, confirming the clinical observations. Large numbers of coccidia, however, were found in all the samples (fig. 2).

Fecal specimen:

1—130 oocysts per low-power field
2—300 oocysts per low-power field
3—A large number of oocysts but too much flocculent débris to permit count
4—100 oocysts per low-power field—heavy film of débris
5—100 oocysts per low-power field

In December, 1926, Doctor E. A. Benbrook, of the Department of Pathology, found coccidia in the feces of a pig by means of the sugar-flotation method. A number of scrapings were made from the small intestine and examined in wet preparations. In one instance 5 or 6 coccidial forms were seen in the epithelial cells. Histologic sections from this material did not show coccidia in the lumen or intracellularly.

In addition to the above herds studied in connection with a survey and study on swine diseases, various groups and individual swine from a number of sources were examined for coccidial infection with a view to determining its prevalence. They all came from within a radius of ten miles from Ames, Iowa.

TABLE I—*Data on other herds examined*

GROUP	PIG	SIZE OR AGE	DATE	NUMBER OOCYSTS	REMARKS
I	1	250 lbs.	Nov. 1	5 per low-power field	Butchered
II	2	250 lbs.	Nov. 1	5 per low-power field	Butchered
	3	250 "		5 per low-power field	Butchered
III	4	150 lbs.	Nov. 14	1 in four fields	
	5	150 "		None	
	6	150 "		1 in three fields	
IV	7	35 lbs.		None	
V	8	300 lbs.		None	Butchered
	9	300 "		5-7 per low-power field	Butchered
VI	10 to 19	35 lbs.		Few oocysts in each sample at different times	
VII	20	60 lbs.		Few in preparation	
	21	60 "		Few in preparation	
	22	60 "		Few in preparation	
	23	60 "		None	
VIII	24 to 38	60 lbs.		Few isolated oocysts	Group raised on clean ground where hogs not permanently kept
	39 to 60	60 "		None	
IX	61	7 months	Nov. 21	3 per low-power field	
	62	7 "		2 per low-power field	
	63	7 "		3 per low-power field	
	64	7 "		3 per low-power field	
	65	7 months	Nov. 27	1 in preparation	
	66	7 "		None	
	67	7 "		None	
	68	7 "		5 in preparation	
	69	7 "		None	
	70	7 "		None	
	71	7 months	Nov. 28	4 in low-power field	
	72	7 "		Few isolated	
	73	7 "		1 in 5 fields	
	74	8 months	Dec. 19	None	
	75	8 months	Dec. 20	1 in 3 fields	
	76	8 "		None	Heavy surface film
	77	8 "		Few in preparation	Heavy surface film
	78	8 "		1 in 3 fields	Average size oocysts 15x17μ
	79	8 "	Dec. 20	2 in preparation	

TABLE I—*Data on other herds examined—Continued*

GROUP	PIG	SIZE OR AGE	DATE	NUMBER OOCYSTS	REMARKS
	80	8 months			This subject was smaller than others of the group and showed a diarrhea. At autopsy, material taken from the rectum contained three large oocyst-like forms 30x36μ Colon—few isolated oocysts Cecum—1 oocyst in each low-power field. Average size 19x24μ Ileum—2-3 oocysts in each low-power field Jejunum—1 oocyst in every 2 fields Duodenum—1 oocyst in every 2 fields. Same size as those found in cecum Stomach—negative The findings as to the number of oocysts in the different portions of the gastro-intestinal tract of this pig were different from the findings described in other parts of this report and will be considered later. Suipestifer agglutinins were absent in the blood of this pig.
X	81				December 1928—Composite specimen of swine feces from a premise where an acute outbreak of bovine coccidiosis prevailed at the time sample was collected. No oocysts were found.

Of the 81 pigs (table I) of different ages from ten different sources and not included in the survey studied herds, 48 were found eliminating oocysts in the feces, while 33 were negative.

CULTURE METHODS

Cultures of oocysts were made from material collected at the various farms where herds were under observation. Some of the sick pigs brought to the laboratory from these herds served as a source of coccidial cultures. Only 5 or 6 samples were generally obtained from such pigs because after being placed in clean pens the number of oocysts passed in the feces gradually decreased. About 100 cc of water was added to 80 grams of feces and thoroughly mixed and strained through a series of copper sieves, 20, 40 and 80 mesh, respectively. To the liquid obtained, potassium dichromate was added. Cultures were made in different ways. Some were placed in large glass dishes, 8 to 10 inches in diameter. Some were kept uncovered, others covered with a glass plate or with a lid belonging to the dish which had an apron about 3 inches overlapping the sides.

Cultivation or development of coccidia by means of a layer of sand covered by filter paper or several layers of filter paper alone in a shallow petri dish was not looked upon with favor, as it necessitates separation of the coccidia from a mass of extraneous material and no attempt was made to concentrate the coccidia in the liquid by centrifuging. Some time ago we experienced

unsatisfactory results with ascaris egg cultures made from material which had been centrifuged. Until such time as we can ascertain the amount of centrifugalization the coccidium can tolerate in its different stages, our cultures will be made directly in potassium dichromate, with or without charcoal. When a dish 8 to 10 inches in diameter is exposed to the air, either in the incubator or in a room, a film is formed on the surface and this, together with dust settling on the surface, permits molds to grow, rendering the cultures useless. A glass cover over the dish or a cotton plug, when a large test-tube or flask is used, inhibits this growth providing the cultures are aerated daily.

Aeration is accomplished by inserting a 0.1 to 1-cc pipette attached to air pressure or a small rubber bulb and passing air through the culture for about two minutes each day. Oxygen is

TABLE II—*Cultures made December 20, 1928*

TUBE	VOLUME (CC)	TEMPERATURE	AERATION	SPORULATION (%) JAN. 4, 1929
1	25	Room	Aerated daily	90
2	50		Aerated daily	90
3	25		Not aerated	0
4	50		Not aerated	0
5	25	Incubator 32°C.	Aerated daily	99
6	50		Aerated daily	99
7	25		Not aerated	5
8	50		Not aerated	3

essential for sporulation and development of coccidia. A temperature of from 21 to 32° C. is favorable for the development of swine coccidia. The depth of the medium does not appear to be a factor provided cultures are aerated. The results obtained in the following set of cultures are representative of those in others. Large test-tubes, 205 x 25 mm., with a capacity of 80 cc, were used. One gram of bone charcoal was added for each 10 cc of culture in 1 per cent potassium dichromate. This brought the medium to a height of 7 mm. in the 25-cc cultures and 13.4 mm. high in the 50-cc cultures.

Beginning with January 4, 1929, tubes 4 and 8 were aerated until January 30. On this date culture 4 (room temperature) showed about 80 per cent sporulated, while culture 8 (incubator) showed about 70 per cent sporulated. More detailed data dealing with cultural results will be included in a later report.

Experimental Series

So far we have not found any pig consistently free from coccidia when fecal examinations were made daily over a considerable period of time by the sugar-flotation method and the preparations thoroughly and systematically examined. Pig 3212, described later, came nearest to being negative. It was negative on weekly examinations from October 14 to December 4. Daily examinations made from December 5 to December 10 resulted in five negative and one positive finding, on December 6 one oocyst being present. It is significant to note, however, that when pigs

Fig. 5. Control pigs from the same source as those shown in fig. 2, but not fed coccidial cultures.

were kept in experiment pens and fecal examinations were made regularly over a period of several months, with only a few isolated oocysts appearing, there never occurred an appreciable increase in the number of oocysts eliminated except when sporulated cultures were fed experimentally. Our observations show further that when field cases passing a considerable number of oocysts are brought to the laboratory and placed in clean, experiment pens, the number of oocysts eliminated gradually decreases until negative findings occur on some days, while on others one or two oocysts may be found if preparations are carefully searched.

On November 14, 1928, two 12-week-old Tamworth pigs (3208 and 3114), weighing about 35 pounds each, were placed in a clean, cement-floored pen of a new barn in which no live stock had ever been kept. Fecal samples removed from the rectum gave the following results with the sugar-flotation method:

3208—1 oocyst found in entire preparation
3114—3 oocysts found in entire preparation. (Ascaris ova absent in both)

FIG. 6. Pigs 3212 and 3215, fed coccidial cultures of swine origin. Note the marked emaciation.

On November 16, 1928, a large composite fecal sample collected from this pen showed only a few isolated oocysts. About 200 cc of a sporulated culture was fed in milk, both subjects being fed from one trough.

On November 17, 1928, composite fecal—same as previous day.

On November 19, 1928, both pigs were indifferent to their surroundings, preferring to remain in the straw. A marked diarrhea was present. A composite fecal sample showed a large number of epithelial cells but no oocysts. Some large forms simulating macrogametes were present.

TABLE III—*Examination of composite fecal samples of pigs 3208 and 3114*

DATE	MATERIAL EXAMINED	NUMBER OF OOCYSTS
1928		
Nov. 22	Composite fecal	6 in low-power field
Nov. 23	Composite fecal	224 in low-power field
Nov. 24	Composite fecal	200 in low-power field
Nov. 25	A—single hard pellet	112 in low-power field
	B—yellow diarrheal sample	160 in low-power field
	C—semisolid	160 in low-power field
	D—yellow, firm, bulky	48 in low-power field
	E—composite sample, great variations in size of oocysts. Both subjects beginning to lose flesh and condition. Haircoats rough and dry	112 in low-power field
Nov. 26	Composite fecal	136 in low power field
Nov. 27	Composite fecal. Some forms small, thin walls	96 in low power field
Nov. 28	Composite fecal	80 in low power field
Nov. 30	Composite fecal	64 in low-power field
Dec. 1	Composite fecal	64 in low-power field
Dec. 2	Composite fecal	80 in low-power field
Dec. 3	Composite fecal	64 in low-power field
Dec. 4	Composite fecal	1 in low-power field
Dec. 5	Composite fecal	5 in low-power field
Dec. 6	Composite fecal	4 in low-power field
Dec. 7	Composite fecal	1 in five fields
Dec. 8	Composite fecal	15 in low-power field
Dec. 9	Composite fecal	1 in five fields
Dec. 10	Composite fecal	1 in five fields
Dec. 11	Composite fecal	1 in six fields
Dec. 12	Composite fecal	1 in low-power field
Dec. 13	Composite fecal	1 in low-power field
Dec. 14	Composite fecal	1 in low-power field
Dec. 15	Composite fecal	1 in five or six fields
Dec. 16	Composite fecal	1 in low-power field
Dec. 17	Composite fecal	1 in ten fields
Dec. 18	Composite fecal	1 in five fields
Dec. 21	Composite fecal	3 in preparation
Dec. 24	Both pigs showed signs of improvement during the past 10 or 12 days. Pig 3214 died, in a fair state of nutrition. The intestines were swollen and the cecum and large colon especially showed thickened walls. The contents were slightly adherent to the mucous membrane. No sections were taken from this pig owing to decomposition	
Dec. 28	Pig 3208	2 in preparation
Dec. 31		1 in five fields
1929		
Jan. 2		1 in six fields
Jan. 3		1 in five fields
Jan. 7		8 in low-power field
Jan. 12		1 in low-power field
Jan. 18	Moved to another pen where no experimentally fed pigs had been kept	5 in preparation
Jan. 20		2 in preparation
Jan. 22		3 in preparation
Jan. 23		2 in preparation
Jan. 25		2 in preparation
Jan. 29		None
Jan. 30		None
Feb. 2		None
Feb. 4		None
Feb. 6		None

FIG. 7. Pigs 3212 and 3215, fed coccidial cultures of swine origin. Prominent pot bellies also developed in the other experimentally fed subjects.

On November 21, 1928, composite fecal showed only two oocysts in entire preparation. The diarrhea had subsided but the pigs refused to leave the litter.

During the past three weeks, pig 3208 presented much improvement over its condition shown several weeks after the culture of sporulated oocysts was fed. However, it has been stunted and

TABLE IV—*Pig 3217*

DATE		NUMBER OF OOCYSTS
1928		
Nov. 14		None
Nov. 29		None
Nov. 30		1 in six fields
Dec. 1		5 in preparation
Dec. 4	On this day there was fed about 130 cc of of a coccidial culture made from material obtained from herd A. About 95 per cent of the parasites in the culture were sporulated at the time of feeding	2 in preparation
Dec. 5	No oocysts were found upon fecal examination. Not all of the culture was consumed by this pig, so warm milk was added the following day to the portion remaining in the trough.	
Dec. 6	This pig did not consume more than about one-third of the dose offered	
Dec. 7		None
Dec. 8		None
Dec. 9		None
Dec. 10		None

shows a distended abdomen and a haircoat that is not so clean and smooth as the control animals shown in fig. 5.

On November 13, 1928, two more Tamworth pigs in good condition were placed in separate, clean pens for fecal study and subsequent experimental feeding of coccidia of swine origin.

Pig 3217 died during the night of December 10-11, 1928. Autopsy showed areas of chronic hepatized pneumonia. The small intestine, especially the ileum, was swollen and congested. After the contents of the cecum were removed and the mucosa

TABLE V—*Pig 3212*

DATE		NUMBER OF OOCYSTS
1928		
Dec. 5	Diarrhea	None
Dec. 6	Feces semisolid, gray color	1 in preparation
Dec. 7		None
Dec. 8		None
Dec. 9		None
Dec. 10		None
Dec. 11		20 in low-power field
Dec. 12		30 in low-power field
Dec. 13		80 in low-power field
Dec. 14		80 in low-power field
Dec. 15		96 in low-power field
Dec. 16		100 in low-power field
Dec. 17		72 in low-power field
Dec. 18		40 in low-power field
Dec. 19		30 in low-power field
Dec. 20		54 in low-power field
Dec. 21		25 in low-power field
Dec. 22		25 in low-power field
Dec. 23		10 in low-power field
Dec. 24		10 in low-power field
Dec. 26		10 in low-power field
Dec. 28		20 in low-power field
Dec. 31		10 in low-power field
1929		
Jan. 2		1 in four or five fields
Jan. 3		1 in four or five fields
Jan. 4		1 in four or five fields

washed off, a fibrinous film remained. No gross hemorrhages were present. Direct smears made from various portions of the intestinal tract did not reveal oocysts, but some large forms simulating trophozoites were noted.

On October 14, 1928, pig 3212, weighing about 35 pounds, was placed in a clean pen. From October 14 to December 4, 1928, no oocysts were found. On December 4, 1928, a culture of sporulated oocysts made from premise A was fed. The entire culture was consumed.

Pig 3212 died on January 5, 1929. The clinical picture and condition of this subject were identical with those of 3215, described later. Pig 3212 appeared most unthrifty, beginning about 20 days after receiving the culture. During this period it showed marked emaciation. The muscles of the loin and gluteal region had wasted so that the prominences of the pelvic bones showed. An outstanding feature in this and all the other experimental cases (except the one which died on the sixth day) was

TABLE VI—*Pig 3215*

DATE		NUMBER OF OOCYSTS
1928		
Dec. 13		1 in preparation
Dec. 14		1 in five fields
	From December 15 to 18 inclusive, this pig was given daily in the feed about 40 cc from different sporulated cultures of swine origin. These cultures varied in the number of coccidia per volume and in the percentage of sporulation. No definite counts were made nor was the actual number of coccidia fed in these preliminary trials determined	
Dec. 15		None
Dec. 16		5 in preparation
Dec. 17		1 in ten fields
Dec. 18		1 in five fields
Dec. 19		25 in low-power field
Dec. 20		2 in entire preparation
Dec. 21		5 in low-power field
Dec. 22		10 in low-power field
Dec. 23		9 in low-power field
Dec. 24		20 in low-power field
Dec. 26		16 in low-power field
Dec. 28		3200 in low-power field
Dec. 29		64 in low-power field
Dec. 30		2000 in low-power field
Dec. 31		96 in low-power field
1929		
Jan. 1		208 in low-power field
Jan. 2		64 in low-power field
Jan. 3		72 in low-power field
Jan. 4		24 in low-power field
Jan. 5		6 in low-power field

the marked emaciation together with an extremely large pot belly and arched back (figs. 6 and 7), giving the pig the appearance of an opossum. The cecum and large colon were filled and distended with feed, the walls being thickened. The wall of the large gut was more than double that of a normal wall. This was based upon actual comparisons with the intestines of two pigs dead of other causes. The contents were quite adherent. All these alterations were not so advanced as in pig 3215, which died

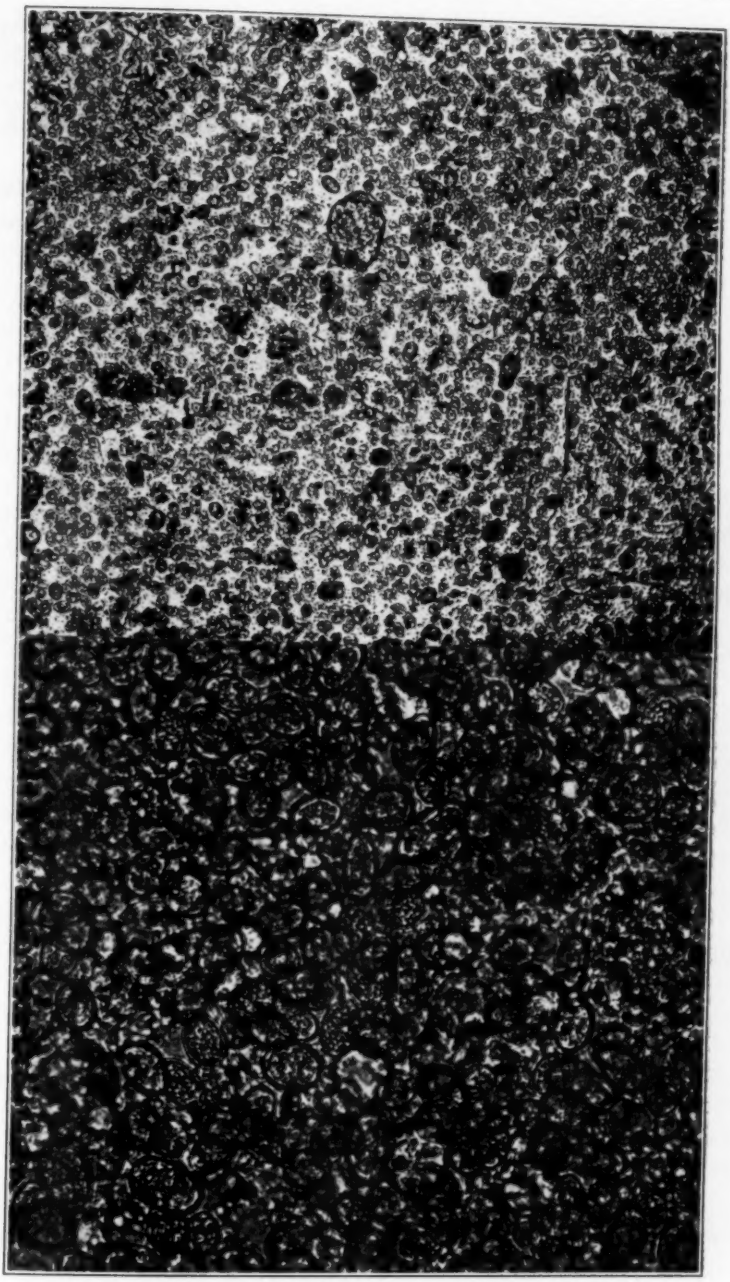

FIG. 8. (Above) From a fecal preparation from pig 3215. Photographed from a thin field after firm pressure on the cover-glass to permit focus. x 100.

FIG. 9. (Below) Fecal preparation from pig 3215 after pressure on cover-glass to permit focus. x 400.

during an earlier stage of the infection. Pig 3212 had improved slightly and had regained some weight during the past ten days although it still presented a characteristic picture (figs. 6 and 7). No suipestifer organisms were recovered from the tissues of this subject.

On December 13, 1928, two more pigs, weighing about 40 pounds and in good condition, were placed in separate pens for experimental feeding and fecal studies.

The oocysts were so numerous that they gave the appearance of the frequently encountered pellicles of light extraneous material coming to the surface in some centrifuged sugar-flotation preparations. Many of these were located on different planes of the microscopic field and undoubtedly some were overlooked in making the count from the high-power magnification (x400). The oocysts were so numerous that it was impossible to obtain a photomicrograph. The cover-glass was pressed down permitting a focus in several of the thinner areas (figs. 8 and 9). From this same centrifuged preparation two more harvests were taken and slides prepared (fig. 3). These show the different sizes and shapes.

Pig 3215 was found dead on the morning of January 6, 1929.

Resume of postmortem: Extreme emaciation, especially pronounced in posterior region with pelvic bones prominent (figs. 6 and 7). Abdomen, greatly swollen, giving the pig a pear-shaped appearance when viewed from above. Lungs, no pneumonia, hypostatic congestion on the left side. Stomach, diffuse cooked appearance of mucosa. Surface easily scraped off. Small intestine, marked catarrhal inflammation. Hyperemia with large quantities of mucus on the surface. The wall was considerably thickened. Cecum and large colon, the wall was very thick, presenting a light and dark gray mottled appearance. Fine granulations indicative of a previous peritonitis were present. On this gray mottled background the blood-vessels anastomosed, giving the appearance of the colored threads in paper currency. The consistency and external appearance of the large intestine suggested an advanced necrotic enteritis specimen. Upon opening this intestine the contents were found evenly glued to the mucous membrane by means of a mucofibrinous exudate, resembling a carpet. After this was removed, the wall was found to be about twice as thick as a subject dead of another condition. Sugar-flotation preparations made from the contents of various portions of the gastro-intestinal tract were as follows:

Stomach—0
Duodenum—0
Jejunum—0
Ileum—0
Colon—2 in low-power field
Rectum—2 in low-power field

Resume of postmortem: This pig became greatly emaciated with hip bones and tuber ischii protruding. Abdomen, tremendously distended and back arched. As viewed from above, it was pear-shaped, like the other subjects. During the last week this pig gained its feet and walked with difficulty. The post-

TABLE VII—*Pig 3216*

DATE		NUMBER OF OOCYSTS
1928		
Dec. 13		None
Dec. 14		1 in low-power field
	Fed daily about 40 cc of different cultures of coccidia of swine origin, December 15 to 18 inclusive	
Dec. 15		1 in five fields
Dec. 16		None
Dec. 17		None
Dec. 18		1 in five fields
Dec. 19		85 in low-power field
Dec. 20		20 in low-power field
Dec. 21		20 in low-power field
Dec. 22		20 in low-power field
Dec. 23		35 in low-power field
Dec. 24		64 in low-power field
Dec. 26		80 in low-power field
Dec. 28		64 in low-power field
Dec. 30		96 in low-power field
Dec. 31		30 in low-power field
1929		
Jan. 1		24 in low-power field
Jan. 2		32 in low-power field
Jan. 3	Pig found dead in the morning.	

mortem changes were identical with those found in pig 3215. The walls of the cecum and large colon were even more thickened and stiff than in pig 3215. As viewed from the exterior they suggested an intestine from an advanced necrotic enteritis subject. No gross caseated membrane was found. The mucosa was covered by a diffuse coat of adherent contents with evidence of desquamation and possibly some shallow necrosis. Congestion was not associated with this process at the time of autopsy. The *S. suipestifer* was not isolated. Sugar-flotation preparations made from various parts of the gastro-intestinal tract were as follows:

Duodenum—0 oocysts.
Jejunum—0 oocysts.
Ileum—6 in preparation. Several large forms similar to trophozoites
Cecum—3 in low-power field
Large colon—30 in low-power field
Gall-bladder—0

In some of the experimentally induced cases of coccidiosis, twelve days subsequent to the first appearance of significant numbers of newly formed oocysts, the number eliminated decreased to a point smaller than is found in many pigs studied in the field. In the first two pigs emaciation did not appear until about four days after the elimination of oocysts had begun, i. e., eleven days after the feeding of the sporulated culture. By the time emaciation had become advanced, the elimination of oocysts had decreased to a number that did not bear a direct relationship to the severity of the infection. In other words, by the time the symptoms were first recognized in the first two pigs, the peak of the oocyst elimination was passed. The length of these periods may vary. If the direct smear method were used, the periods of oocyst elimination would be shortened because the sugar-flotation method will demonstrate small numbers of oocysts or ova which cannot be found by the direct smear. These facts are of paramount importance in the diagnosis of coccidiosis in the field. If pigs were subjected to a short heavy exposure of sporulated oocysts, the attention of the diagnostician would not be called to such a herd until the symptoms were advanced. If at such time a fecal examination were made, the number of oocysts found in the feces may be insignificant. Should the ingestion of sporulated oocysts extend over a long period, then newly formed, non-sporulated forms would be present in the feces and a diagnosis could be made readily. In one field study, sporulated oocysts were recovered in the mud of a hog-wallow. A very heavy rainfall preceded our next visit to this farm and when samples were collected it was noted that nearly all the loose soil in this wallow had washed away, leaving a heavy bed of fibrous residue of corn stalks and other plant material. Liberal quantities of this material were examined but no oocysts could be demonstrated. This indicates that the stage of the infection encountered at the time of examination, as well as the circumstances under which examination is made, are important. We had similar experiences with coccidiosis in chicks. A number of

times, in outbreaks of coccidiosis, chicks showing marked emaciation were autopsied. In some the oocysts were numerous, while in others from the same lot, equally emaciated, the oocysts were scarce. These experiences lead to the belief that the diagnosis of coccidiosis in swine is a problem that should be given

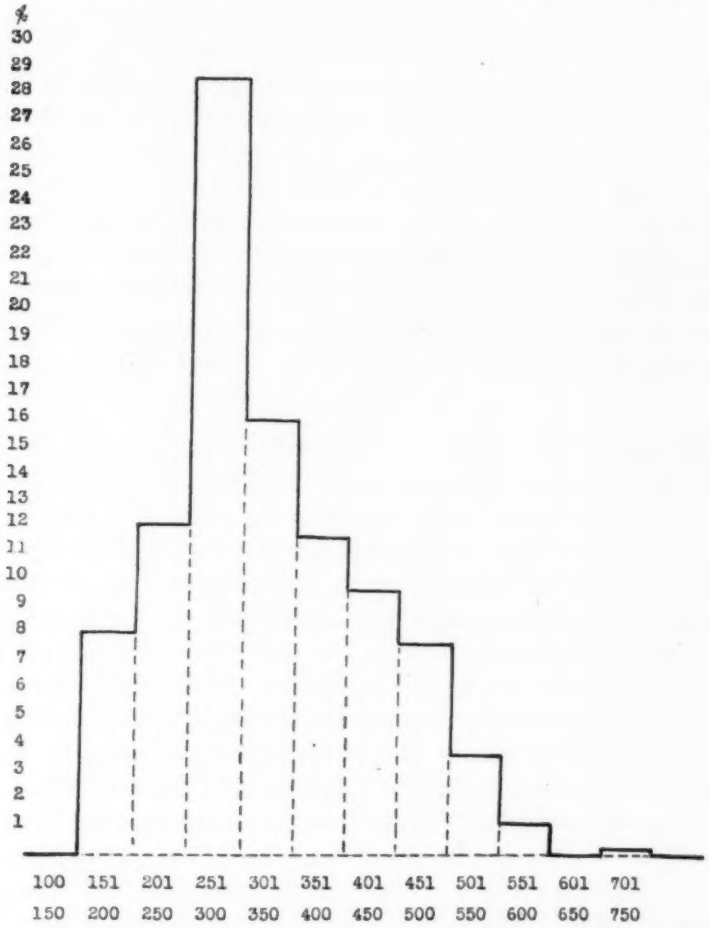

FIG. 10. Chart showing 300 oocysts of swine origin, grouped according to size as determined by their circumscribed rectangles, indicated in square microns by numbers under the base line.

careful consideration based upon further detailed studies from all angles.

The experimentally fed pigs began to eliminate newly formed oocysts in the feces in four to seven days after the feeding of sporulated cultures. In another experiment underway, in which

sporulated oocysts of swine origin were fed to four pigs weighing about 80 pounds, newly formed oocysts appeared in the feces of one subject seven days later and in the other three were found in large numbers six days after feeding.

Pathogenicity of Coccidia from Swine

A difference of opinion prevails regarding the pathogenicity of coccidia in swine. Most of the investigators who found oocysts in slaughter-house swine minimize the seriousness of this condition because they found no gross alterations, although Cauchemez[16] calls attention to a possible severe pathogenicity in young pigs. Several observers attribute extensive losses in young pigs to infection with coccidia. Our own investigations show that marked clinical manifestations and pathological alterations of the large intestine are produced by the ingestion of sporulated oocysts of swine origin. The possibility of apparently normal hogs which eliminate oocysts being carriers must not be ignored.

Size

We believe the Eimeria encountered by us in swine to be identical with that described under the designation of *Eimeria debliecki* (and synonyms) by European investigators. Krediet's studies indicate that only one species of coccidia is present in swine if size is the criterion. By grouping the oocysts according to their volume, expressed in cubic microns, he showed that they belonged to one species with medium-size forms predominating, both extremes in size being connected by intermediate groups. Our studies bear out Krediet's findings. Three hundred oocysts from ten different sources (swine) were plotted as to size on the basis of their circumscribed rectangles (fig. 10). In most instances the measurements were made from photomicrographs (x400) and the oocysts used in the determination are representative forms with regard to size. It will be noted that the largest number belong to the middle groups and both extremes are connected by intermediate sizes as in Krediet's report. In addition to the 300 oocysts plotted, two forms were encountered measuring 46μ by 38μ and 32μ by 28μ, 1748μ^2 and 896μ^2 respectively. At the time these two forms were noted, a question was raised as to whether or not they were oocysts. The average length of the 300 oocysts was calculated to be 20$\frac{2}{3}\mu$ and the average width 15$\frac{1}{2}\mu$, while their extreme measurements were as follows:

Minimum		*Maximum*	
Length	Width	Length	Width
12μ	12μ	32μ	28μ

Table VIII shows the average length and width of each one of the ten groups of oocysts comprising the 300 forms upon which the chart (fig. 10) is based.

The marked variations in size between some of the groups of swine oocysts would indicate that other factors in addition to size must be considered in the identification of coccidia of swine origin.

Coccidia of Bovine Origin

The identification of coccidia of swine origin is further complicated by the results of an experimental coccidial infection produced in swine by means of coccidia of bovine origin. During the

Table VIII—*Average length and width of oocysts*

Swine Group	Oocysts Measured	Average Length (μ)	Average Width (μ)
1	50	23	17
2	101	20.5	14.8
3	12	23.8	18
4	6	24	19
5	64	19	14.8
6	5	18	14.8
7	5	16.6	14.4
8	44	19.6	14.9
9	6	23.8	19
10	7	24	18.5

latter part of December, 1928, cultures of oocysts were made from an outbreak of bloody diarrhea in calves caused by coccidia. After sporulation these cultures were fed to two pigs weighing about 80 pounds each. Six days after feeding, there appeared in the feces of the fed pigs a large number of newly formed non-sporulated oocysts. The oocysts recovered from the swine feces as a result of feeding coccidia of bovine origin were compared with those of swine origin as to size. From a limited number of measurements made on the bovine forms it was impossible to differentiate coccidia of swine and bovine origin. Riovlta believed the swine forms to be identical with *Eimeria zürni*. Cauchemez[16] states: "The *Eimeria zürni* yields rounded or elongated oocysts whose dimensions resemble those otherwise very variable coccidia of the pig." The inability to differentiate the oocysts obtained from swine and cattle on the basis of size

alone, and the ability to infect swine with coccidia recovered from calves makes the question of identity an important one because of the close contact of swine and cattle in the feed-lot. Further investigations are in progress in this connection and will be reported later.

The possibility of a group relationship in coccidia, such as exists in the Pasteurella and tubercle bacilli groups, must be kept in mind in planning further studies. The degree of pathogenicity of coccidia of bovine and swine origin for other species of hosts also should be considered.

Conclusions

1. Coccidiosis is believed to be an important but heretofore overlooked disease of swine in America.

2. Oocysts of swine origin have been cultured in potassium dichromate with the addition of charcoal. A temperature of 21° C. to 32° C. was found suitable for growth. Daily aeration was found necessary for development.

3. The coccidia of swine origin encountered by us proved to be Eimeria, the oocysts developing four sporocysts, each having two sporozoites.

4. When cultures of sporulated oocysts made from field cases were fed to healthy pigs, an infection resulted.

5. Newly formed non-sporulated oocysts appeared in the feces in from four to seven days after such experimental feeding.

6. The severity of the infection produced is dependent upon the number of sporulated oocysts fed and the condition of such cultures. When cultures are held too long after complete sporulation, they lose their ability to infect, which probably accounts for some of the negative feeding results obtained by some investigators.

7. Based upon size, the forms found in swine of Iowa correspond to *Eimeria debliecki* reported on the Continent. After plotting 300 non-selected forms from ten different sources on the basis of their circumscribed rectangles, we believe that only one species prevails in swine.

8. The sizes of oocysts eliminated by experimental and field cases varied, depending upon the stage of the infection when they were recovered. As a general observation, during the first few days of oocyst elimination, their size fell near the maximum measurements. Later, smaller forms began to appear and the proportion of larger oocysts decreased.

9. If the prevailing conception of the life cycle of coccidia is accepted, these forms were shown to be pathogenic because the newly formed oocysts found in the dejecta were the result of extensive invasion of the intestinal epithelium followed by the sexual multiplication which gives rise to the formation of oocysts.

10. The experimentally fed pigs developed marked clinical manifestations. Bloody diarrhea was not observed, but constipation was noted.

11. The experimentally induced cases of coccidiosis in swine (by means of strains of swine origin) presented a pronounced thickening of the wall of the large intestine, in some cases the wall being twice as thick as those of control subjects, together with a diffuse adherence of the contents. As viewed from the exterior, the intestine suggested an advanced case of suipestifer enteritis, but the heavy caseated membrane was not present.

12. The *S. suipestifer* was not isolated from any of the subjects included in these studies.

13. Coccidia recovered from an outbreak of coccidiosis in calves, associated with bloody diarrhea and deaths, proved to be Eimeria. When sporulated cultures of these oocysts were fed to pigs a definite infection resulted. Six days after feeding such cultures, newly formed non-sporulated oocysts appeared in the feces of the experimentally fed pigs, indicating that epithelial cell invasion had taken place.

14. Size of the oocysts recovered does not appear to be a sound basis for the differentiation or identification of coccidial species. The bovine form could not be distinguished from the swine form on the basis of size alone. Investigations are in progress to determine whether or not *Eimeria zürni* and *Eimeria debliecki* are distinct species or identical and equally pathogenic for both calves and pigs. Possibly there is a group relationship as in the case of the Pasteurella and tubercle bacilli groups.

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Chattanooga Veterinarians Open New Hospital

Drs. F. W. Morgan (McK. '06) and G. P. Hatchett (Ind. '22), of Chattanooga, Tennessee, opened a new small-animal hospital at 1025-27 McCallie Avenue, on November 16. The building is strictly modern throughout and contains accommodations for 55 patients, with offices, library, laboratory, observation room, operating room, bathroom, etc. Ambulance service is included in the appointments. *The Chattanooga Daily Times* of November 18 gave the new hospital a very nice write-up.

Good Publicity

Through the agency of the Upper Iowa Veterinary Association, the Titonka (Iowa) *Topic*, for October 31, 1929, printed in full the address of Mr. J. H. Weiner, Kansas City chemist, delivered before the recent meeting of the Eastern Iowa Veterinary Association, on the subject of stock remedies. In commenting on the activities of the nostrum manufacturers and vendors, Mr. Weiner said that they absolutely disregard the science of therapeutics and pharmacology in the selection of the ingredients for their pharmaceutical monstrosities.

INFECTIOUS PUSTULAR DERMATITIS OF SHEEP AND GOATS

By J. A. HOWARTH, *Davis, California*

University Farm, University of California

Infectious pustular dermatitis of sheep and goats is a disease reported by several writers as affecting these animals in widely separated countries of the world, but, to our knowledge, it has heretofore never been described in the United States. This disease was first brought to our attention in July, 1928, and, since that time, we have had four affected bands under observation. This disease is a highly infectious one in which exanthematous localizations are very characteristic, passing through the stages of macule, papule, vesicle, pustule, and scab formation. The affection is observed in sheep and goats of all ages, is most prevalent in lambs and ewes following lambing, and is caused by a filtrable virus. We propose to accept the name infectious pustular dermatitis of sheep and goats, similar to the title given by R. E. Glover,[1] of Cambridge, England, for our work at this station has conformed to his in the etiology, experimental transmissions, histopathology, and all other details investigated.

DISTRIBUTION OF THE DISEASE

Our attention was first called to this condition in July, 1928, when the disease appeared in a flock of 105 aged ewes, of which 97 became affected. In December, 1928, our attention was called to a second and a third outbreak, which involved 630 ewes with lambs at their side. The latter part of the same month, a fourth band of 520 ewes, with lambs at their side, became affected. In the last three bands, the lambs were the first to show the infection.

In looking up the references in the literature which seemed to describe this condition, we found reports from several parts of the world. Thus Zeller,[2] in 1920, reported his studies in Berlin, on material supplied to him from Southwest Africa by a veterinarian in Atziwarango. In 1922, Blanc, Melanidi and Caminopetros,[3] reported a similar disease in goats in Greece. In 1923, Aynaud[4] published his studies on this condition under the name of "chancre du mouton," and stated that it was a widespread disease in France and was the cause of great loss, especially among lambs that were being prepared for the butcher. In

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1923, Moussu[5] described a seasonal disease under the heading of "contagious ecthyma of the lips of sheep," and stated that the condition occurred chiefly in the summer. Lanfranchi,[6] in 1925, reported the existence in Italy of a similar disease caused by a filtrable virus. Jacatot,[7] in 1925, found a similar condition very common in goats, in Annam, Indo-China, and called it "maladie du chancre."

The above references seemed to exhaust the literature on the disease at this time. After checking our experimental data with the above-mentioned authors, we received in January, 1929, an article written by Sir Arnold Theiler,[8] entitled, "Exthyma Contagiosum of Sheep and Goats," published in October, 1928; and also an article by R. E. Glover[1] under the title of "Contagious Pustular Dermatitis of the Sheep," published in December, 1928.

FIG. 1. Some animals show affections of the outer surface of the face which is more or less free from wool, as the eyelids and the inside of the ears.

These two articles describe a condition which we believe to be identical with the disease with which we have been working.

NATURE OF THE DISEASE

Evidently this disease has been present in California for some time, and has been called by various names, including "doby mouth," "sore mouth" and "pox," by the sheepmen. It is an infectious, exanthematous affection of the skin, caused by a filtrable virus. The affection, observable in sheep of all ages, is most prevalent in lambs and ewes following lambing. The condition tends to assume an enzootic form. In one band of 105 aged ewes, 97 became affected; in two other bands of 380 ewes and 250 ewes, respectively, both with lambs at their side, practically every animal became affected. The disease was first noticed as

affecting the exterior of the lips, especially the commisures, and the nostrils. Later, the ewes showed lesions on the udder, teats, and inside of the thighs. In this case, the virus already located in the lesions on the lips and nostrils was most probably transmitted to the udder and teats either by licking or from an affected lamb nursing.

No mortality nor secondary complications have been observed during these investigations. According to Jacotot, such complications may arise, causing a fifty per cent mortality because of secondary lesions accompanied by suppuration. Aynaud, also,

FIG. 2. Some of the lambs show lesions confined to the exterior of the lips, these lesions becoming confluent and forming a wart-like protuberance. If the crusts are removed, a bleeding, elevated surface results.

states: "A mortality of ten to twenty per cent may occur when secondary pulmonary lesions ensue, especially when the flock is in low condition." We have observed in the condition of the lambs a considerable loss resulting from soreness of the mouth which produces a lack of a desire to graze, and, also, from a decrease in the amount of milk they receive, when the ewes try to prevent them from nursing. In many cases where the teats and udders were badly affected, the ewes would disclaim their lambs, the retention of the mammary secretions resulting in mammitis.

Symptoms

The first lesions are a slight reddening and elevation of the skin, which loses its color on pressure. This is followed by the development of vesicles from the papular stage, rising above the surface of the skin with a small depression in the center. The skin around the vesicles is most generally hyperemic, with a slight edema of the tissues. As the vesicles increase in size, their contents become purulent and advance into the pustular stage, which ruptures, allowing a white cloudy lymph to a yellowish exudate to be exuded. Later a yellowish-brown crust or scab

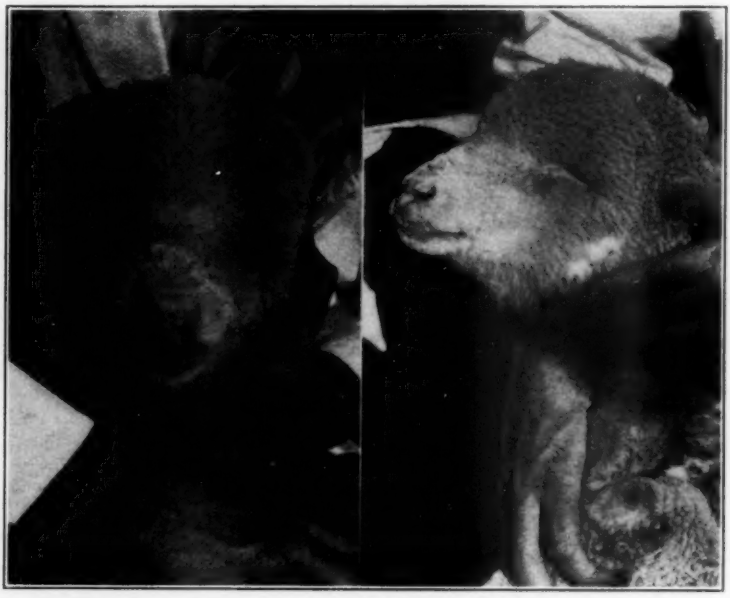

Fig. 3. The same animal as shown in figure 2, thirty-one days later. This animal received no treatment. The lesions all healed, leaving no scars or signs of any previous injury to the tissues.

forms, which gradually becomes very dark and drops off by the twenty-fourth day, leaving no scar.

The lesions in lambs are usually located on the lips, nostrils, and neighboring tissues, forming thick crusts or scabs, but, in the more serious cases, may extend into the mouth, affecting the tongue, the gums, and the roof of the mouth. Some animals show affections of the outer surface of the face, which is more or less free from wool, as the eyelids and the inside of the ears. Occasionally lesions are found under the base of the tail. Some of the lambs at first showed lesions confined to the exterior of

the lips. These lesions were in the form of thick crusts or scabs, later becoming confluent and forming a wart-like protuberance. If the crusts are removed, a bleeding, elevated surface results.

Continual rubbing and scratching may make the vesicles and pustules hard to observe on the lips and nose. Secondary invaders gain entrance and produce a greater inflammation of the part, followed by edematous swellings and necrosis. Sometimes the presence of a disagreeable odor might tend to mislead one into making a field diagnosis of a necrophorus infection.

In ewes, the mammary glands show more typical lesions because of the protection afforded them by their location and

FIG. 4. The mammary glands of a ewe, showing vesicles, pustules and scab formation.

because the ewes so affected try to prevent the lambs from nursing. Vesicles and pustules often are found on the inside of the thighs, and, in some cases, have extended down to the hocks.

The histology of the lesions has been worked out very comprehensively by Glover, of Cambridge; he divides it thus into three stages:

> First stage. (Papulo-vesicle.) * * * The earliest change to be noted is a commencing proliferation of the cells of the rete Malpighii. * * * The vesicles arise between the more superficial cells immediately beneath the stratum lucidum and are represented by collections of polynuclear leucocytes. * * * Second stage. (Vesico-pustule.) * * * The cells in this area are undergoing still further degeneration, forming an

irregular network which bears a strong resemblance to the "Ballonisante" stage seen in the lesions of vaccinia. * * * The vesicles have now become considerably larger, and, as the lesions become older, the migration of leucocytes continues until the vesicle changes to a pustule containing a debris of epithelial cells and leucocytes with a few micrococci. * * * Third stage. (Scab.) * * * During this phase, the pustule enlarges and finally breaks through the stratum lucidum. The crusts are formed from the cellular debris which is thus liberated, fibrin, and the remains of the stratum lucidum. In some places, owing no doubt to the more intensive invasion of the breach thus formed by the various bacteria, the epidermis is completely disorganized. The crusts then cover a mass of epithelial cells in various stages of degeneration, intermixed with polynuclear leucocytes and young fibroblasts. Healing now takes place beneath the crusts by regeneration of the epithelium and gradual resorption of the cell debris.

Differential Diagnosis

Our attention was particularly given to establishing the distinct differences between this affection and those conditions supposedly caused by the *Bacillus necrophorus* organism, especially lip and leg ulceration. Melvin and Mohler,[9] in 1910, published an article entitled, "Lip and Leg Ulceration of Sheep (Necrobacillosis)," and described a very contagious form of sore mouth in lambs, usually seen during the fall of the year, although observed in some cases earlier in the season. The lesions described in this condition are very similar to infectious pustular dermatitis. In this article they state:

In such lesions, the quiescent coccoid and bacillary forms of the bacillus will predominate, while only an occasional short filament will be observed. . . . It is not, however, the feed or the pasture or the fact that they have just been weaned which of itself causes the lesions; but, in addition to these predisposing causes, the necrosis bacillus becomes present, and the disease continues to spread.

Wallis Hoare[10] describes a disease, "contagious pustular dermatitis of sheep, caused by the bacillus of necrosis," and, under this caption, he uses the following synonyms: malignant aphtha; contagious dermatitis; "orf"; lip and leg ulceration of sheep; crusta labialis; ulcerative stomatitis; red foot; contagious ecthyma; "hair and hoof"; "mouth and foot"; "carbuncle of the coronary band"; necrobacillosis of the sheep. French: la maladie ulcereuse des levres et des pattes du mouton; mal de bouche. He described four types of this affection similar to those set forth in the report of Melvin and Mohler. First, that type attacking the lips and the legs; second, the venereal type, affecting the genital organs in both sexes; third, the type known as "red foot" or foot rot; and fourth, "mouth disease" (mal de bouche).

From a study of over 1550 cases, it seems evident that we are not dealing with the disease described by the two above-mentioned authors, but with a new clinical entity. We were unable

to isolate *Bacillus necrophorus* from any of the cases affected with infectious pustular dermatitis of sheep and goats examined at this station.

The following differential characteristics are given:

TABLE I—*Differential diagnosis between lip and leg ulceration of sheep and other diseases caused by Bacillus necrophorus*

	BACILLUS NECROPHORUS	FILTRABLE VIRUS
Susceptibility of various animals	Horses, cattle, sheep, goats, dogs, hogs, rabbits, chickens, mice, guinea pigs, monkeys, kangaroos, reindeers, etc.	Sheep, goats
Ages	All ages	All ages, usually young lambs, and ewes following lambing
Course and duration	Slow and protracted, individual sheep, several months or more; flocks, 8 or 10 months to over a year	Rapid; individual sheep, approximately 6 weeks; flocks, 2 to 3 months
Period of Incubation	3 to 10 days, occasionally 15 days	4 to 7 days
Parts affected	Skin, lips, mucous membranes of mouth and upper air-passages; lungs, digestive tract, navel, abdominal viscera, hoofs, cartilage, bone, muscles and joints, mammary gland, teats; in fact, no tissue seems to be immune to the invasion of *Bacillus necrophorus*	Exterior of lips, gums, palate, portion of the face free of wool such as the eyelids, and inside of the ears; mammary glands, teats, posterior part of abdomen and inside of thighs which are free from wool
Lesions or histopathology	Progressive tissue necrosis characterized by caseous degeneration and invasion of the deeper tissue. Diphtheroid lesions of the mucous membranes. Abscess formation in the connective tissue, liver or lungs	Slight reddening and elevation of the skin; papules, vesicles, pustules, and scab formation
Intravenous injection	Followed by metastatic abscesses especially the liver and lungs	No lesions produced
Immunity	None	Definite

BACTERIOLOGICAL INVESTIGATIONS

When the infection was first encountered, in July, 1928, both aerobic and anaerobic cultures were made from the lesions. The inoculum for these cultures was obtained by carefully lifting the membrane from the vesicles, raising the scabs with sterile forceps, and applying the platinum loop to the purulent material beneath. This inoculum was placed on culture media consisting of plain

agar, blood-agar, serum-agar, and gentian-violet agar. Anaerobic conditions were obtained by stab culture and by surface cultures placed in Novy jars, and the oxygen was exhausted by pyrogallic acid and sodium hydroxid. Pure cultures of what proved to be *Staphylococcus albus* were obtained in the aerobic tubes; nothing of interest developed anaerobically. The gentian-violet-agar cultures all remained sterile. These cultures were made from material taken from pustules in order to eliminate as much as possible secondary invaders found in the older scab lesions.

With material taken from the lesions, pocket inoculations were made into four rabbits to ascertain whether or not *Bacillus necrophorus* was present. At the point of inoculations, local abscesses developed. Material taken from these abscesses was examined and found to be *Staphylococcus albus*. At the end of thirty days, all of the rabbits were alive and normal. One was autopsied and no lesions found.

The cultures of *Staphylococcus albus*, and also purulent material taken from pustules on the affected animals, were inoculated separately upon three test lambs (10, 12 and 15, table II) by scarification of the skin on the posterior part of the abdomen which was free from wool. In all cases, pustules developed in from 8 to 24 hours; but these lesions were not characteristic of the natural infection. The pustules coalesced, ruptured and healed in three to four days. Two of these animals (10 and 12), inoculated with the purulent material, developed typical lesions of the disease, beginning on the sixth and seventh days, respectively, these lesions passing through the papule, vesicle, pustule and scab stages. The scabs dropped off by the 24th day. This series of experiments eliminated *Bacillus necrophorus* and *Staphylococcus pyogenes albus* as the causative factors in this disease.

On November 6, 1928, our attention was called to a second and a third outbreak in two flocks of sheep on adjoining lands, approximately ten miles distant from the animals studied in July. A scab from the face of an affected young lamb was again rubbed into a scarified area on the posterior part of the abdomen of lamb 18. (See table II.) Pustules again developed in twelve hours, and complete healing took place in three days; but, on the fourth day, a slight reddening was noticed, followed on the sixth and seventh days by vesicles and pustules. The latter ruptured on the twelfth day, and scab formation followed, which dropped off on the 23rd day. In this case, as in that of the two previously inoculated animals (10 and 12), there was not so

TABLE II—*Inoculation of lambs*

LAMB	DATE (1928)	INOCULUM	DILUTION	POINT OF INOCULATION	FIRST APPEARANCE OF LESIONS CAUSED BY STAPHYLOCOCCUS	FIRST APPEARANCE OF LESIONS CAUSED BY A FILTRABLE VIRUS	REACTION	REMARKS
10	9–22	Material from pustules in pyhs. NaCl solution	1–1,000 1–10,000	Right side Left side	Pustules 12 to 24 hours	6 to 7 days	+ +	Characteristic lesions starting on 6th to 7th day
12	9–22	Material from pustules in phys. NaCl solution	1–1,000 1–10,000	Right side Left side	Pustules 12 to 24 hours	6 to 7 days	+ +	Characteristic lesions starting on 6th to 7th day
15	9–22	Culture of *Staphylococcus albus* from pustule	1–1,000 1–10,000	Right side Left side	Pustules 12 to 24 hours	Negative	— —	Pustules dried up on 3rd to 4th day. No characteristic lesions
18	11–8	Scab	1–1,000 1–10,000	Right side Left side	12 to 24 hours	6th day	+ +	First lesions disappeared on 4th day. Characteristic pustules developed on 6th to 7th day

serious a spreading of the infection as occurred in the naturally infected cases.

The study of the literature, together with that of the manifestations in the experimentally inoculated animals, led to the thought that the primary cause of the infection might be a filtrable virus.

On December 25, 1928, a fourth band became affected, about fifteen miles from the second and third affected flocks. On January 3, 1929, material was obtained from ewe 2, which had a natural attack of the disease. The membranes covering the vesicles were carefully lifted with sterile forceps, and a platinum loop was applied to the purulent material. This material was placed in a sterile mortar and enough distilled water added to make a paste; it was then thoroughly ground and an addition of 50 per cent glycerin containing a buffer of KH_2PO_4 (reaction pH 7.5) was incorporated with it. With the assistance of Dr. J. Traum, three guinea pigs were inoculated with the virus in the pads of the hind feet by scarifying and "tunneling" the epidermis. These tests resulted negatively: no vesicles appeared on the pads of any of the feet, and all of the animals survived the inoculation. On January 7, 1929, material from ewe 2 was inoculated into the inside of the lips and interdental pad of a Jersey cow which was apparently in good health and condition. The part was scarified with a stilet, and the material applied with a sterile swab. No characteristic lesions developed at the seat of inoculation. This animal was kept under observation for one month; no lesions developed, and the animal is now in the best of health.

On January 7, scabs taken from ewe 2 were desiccated over sulphuric acid for 72 hours and emulsified with physiological salt solution. This virus was inoculated into two healthy pigs by scarifying the epidermis on the inside of the thighs. The inoculation of these two animals resulted negatively. On the same day, corneal and epidermal inoculations of two rabbits with the same virus proved negative. A dog was inoculated with the virus by scarifying the epidermis of the inside of the thigh, and the result was also negative. The data on these inoculations are given in table III.

The Effect of Preservatives on the Virus

The scabs obtained from the second and third flocks affected with the disease were finely ground in a sterile mortar. The material was then placed in a desiccator over sulphuric acid for

TABLE III.—*Inoculation of animals other than sheep and goats*

TABLE III—*Inoculation of animals other than sheep and goats*

ANIMAL	DATE (1929)	VIRUS	DILUTION	POINT OF INOCULATION	PERIOD OF INCUBATION (DAYS)	RE-ACTION	REMARKS
G. P. 111	1–3	In 50% glycerin containing KH_2PO_4 (pH 7.5)	1–1,000 1–10,000	Pads of hind feet	—	—	No characteristic lesions developed
G. P. 112	1–3	In 50% glycerin containing KH_2PO_4 (pH 7.5)	1–1,000 1–10,000	Pads of hind feet	—	—	No characteristic lesions developed
G. P. 113	1–3	In 50% glycerin containing KH_2PO_4 (pH 7.5)	1–1,000 1–10,000	Pads of hind feet	—	—	No characteristic lesions developed
Cow 1	1–7	In phys. NaCl solution	1–1,000 1–10,000	Inside of thigh Inside of lip	—	—	No characteristic lesions developed
Pig 161	1–8	In phys. NaCl solution	1–1,000 1–10,000	Inside of thigh	—	—	No characteristic lesions developed
Pig 162	1–8	In phys. NaCl solution	1–1,000 1–10,000	Inside of thigh	—	—	No characteristic lesions developed
Dog 1	1–8	In phys. NaCl solution	1–1,000 1–10,000	Posterior part of abdomen	—	—	No characteristic lesions developed
Rabbit 114	1–8	In phys. NaCl solution	1–1,000 1–10,000	Cornea of eye Inside of thigh	—	—	No characteristic lesions developed
Rabbit 115	1–8	In phys. NaCl solution	1–1,000 1–10,000	Cornea of eye Inside of thigh	—	—	No characteristic lesions developed
Lamb 86 (Control)	1–8	In phys. NaCl solution 50% glycerin	1–1,000 1–10,000 1–1,000 1–10,000	Right side of abdomen Left side of abdomen	5 to 6 6 to 7	+ +	Vesicles and pustules developed on 5th to 6th day. Scab formation followed, dropping off by 24th day

24 hours, next removed, placed in a mortar and ground to a fine powder, and again placed in the desiccator over sulphuric acid for an additional 48 hours. One hundred milligrams of the dried scabs were put in a mortar, and distilled water added to make a paste; enough distilled water was then added to make a 1–100 dilution. The material was shaken for about ten minutes, placed in sealed tubes, and allowed to stand for three hours at room temperature (21°C.). The tubes were then centrifuged fairly slowly for five minutes, and the supernatant fluid was removed. Series of dilutions were made in physiological salt solution, 2 per cent boric acid, 25 per cent glycerin, 50 per cent glycerin, and 50 per cent glycerin containing a buffer of KH_2PO_4 standardized to pH 7.5, as follows: 1–500; 1–1,000; 1–5,000; 1–10,000; 1–50,000; 1–100,000.

In all cases, cultures were made from these dilutions and tested for sterility. These dilutions always were placed under controlled temperature conditions as soon as possible, and were kept at 15° C. throughout the experiment.

Filtration Experiments

Fresh materials from the pustules and dried scabs obtained from a sheep having a naturally contracted case of the disease were emulsified in enough physiological salt solution to make a 1–1,000 and 1–2,500 dilution and thoroughly shaken for about ten minutes to break up the strings of mucus. These dilutions were allowed to stand at room temperature for three hours in sealed tubes. They were then slowly centrifuged for five minutes, after which the supernatant fluid was withdrawn and passed through different types of filters, namely: Seitz asbestos filter, Uhlenhuth type; Chamberland L 2; Mandler No. 7; and Berkefeld N and W. The filtration was carried out at room temperature (21°C.); the actual time required for the operation never exceeded fifteen minutes. During the filtration process, a suction-pump was used, carrying a negative pressure of 15 inches of mercury. Two cc of a 24-hour culture of *Staphylococcus pyogenes albus*, obtained by washing the agar slants with a physiological salt solution, was then added to the liquid before filtration. The filtrates were cultured and found negative at the end of six days. Throughout all these experiments, the filtrates were kept in the ice-box at a controlled temperature of from 10° to 15°C.

Filters through which infectious material had previously been passed were first immersed in a non-coagulating germicide long

TABLE IV—*Experiments with the virus in different menstrua and in different dilutions*

LAMB	DATE	VIRUS IN	POINT OF INOCULATION	DILUTION	PERIOD OF INCUBATION (DAYS)	REACTION	REMARKS
3	12–21–28	25% glycerin	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	7 to 8	+ + + —	Vesicles and pustules 7th to 8th day. Ruptured 12th to 13th day. Scab fell off 24th day
13	12–21–28	50% glycerin	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	7 to 8	+ — — —	Vesicles and pustules 7th to 8th day. Ruptured 12th to 13th day. Scab fell off 24th day
15	12–22–28	50% glycerin containing KH_2PO_4 (pH 7.5)	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	8 to 9	+ — — —	Vesicles and pustules 8th to 9th day. Ruptured 12th to 13th day. Scab fell off 24th day
16	12–22–28	2% boric acid	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	6 to 7	+ + + —	Vesicles and pustules 6th to 7th day. Ruptured 11th to 12th day. Scab fell off 24th day
17	12–22–28	Phys. NaCl solution	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	5 to 6	+ + + —	Vesicles and pustules 5th to 6th day. Ruptured 11th to 12th day. Scab fell off 23rd day
9 (Control)	12–22–28	5% glycerin Phys. NaCl solution	Right side Left side	1–1,000 1–10,000 1–50,000 1–100,000	Negative	— — — —	Animal had previously a natural attack of the disease
3 (Goat)	1–10–29	Phys. NaCl solution 25% glycerin	Right side Left side	1–1,000 1–10,000 1–5,000 1–10,000	6 to 7	+ + + +	Vesicles and pustules 6th to 7th day. Ruptured 11th to 12 th day. Scab fell off 23rd day

enough to produce sterilization. The outside of the candle was carefully cleaned with a brush to remove all adhering material. Water was forced from within outwards under pressure until all visible impurities were removed. The candles were then placed in a two per cent solution of sodium carbonate for one-half hour, after which they were placed in distilled water and boiled for one hour. The candle was then washed from within outwards with water and placed in the autoclave for twenty minutes at 20 pounds pressure. The new filters were washed with distilled water, which was forced through them from within outwards; they were then placed in the autoclave for twenty minutes at twenty pounds pressure.

The filters used in all these experiments were new, with the exception of three candles which were later discarded because the filtrates were not always uniform in producing the disease. The virus of this disease passed through earthen-ware filters, but with great difficulty; it seems highly probable that during the filtration process there was some loss of active materials.

In the following experiment, infective filtrates were used that were obtained by passing through a Mandler filter No. 7 and a Chamberland L 2. (See table VI.)

We were able to demonstrate the presence of a filtrable virus both in the papulo-vesicular lesions and in the dried scabs. Repeated attempts were made to obtain an infective filtrate from different types of filters, but with varying results. The failure of the virus to pass through the same type of filter each time is evidently due to some uncontrollable factors which influence the infectivity of the filtrates; at the same time, if we take into consideration the fact that no two filters are equally permeable, many external circumstances may modify their permeability.

The difficulty encountered in filtering the virus of this disease may be ascribed to the intracellular location of the virus. If such is the case, the filter removes the cells, and, therefore, the greater part of the infecting agent is held within the candle. In this disease, the filtrability of the etiological agent is carried out with the greatest difficulty. At present, the results obtained with the different types of filters are rather confusing, and little can be said with assurance.

Immunity

The duration of immunity in this specific disease should be studied more carefully over a longer period of time before one

TABLE V—*Experiments with filtrates*

LAMB	DATE (1929)	TYPE OF FILTER*	POINT OF INOCULATION	DILUTION	PERIOD OF INCUBATION (DAYS)	REACTION	REMARKS
141	1–11	Seitz	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	Negative	— — — —	No characteristic lesions
142	1–11	Chamberland L 2	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	6 to 7	+ + + —	Vesicles and pustules 6th to 7th day. Ruptured 12th day. Scab fell off by 24th day
143	1–11	Berkefeld N	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	Negative	— — — —	No characteristic lesions
144	1–11	Berkefeld W	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	Negative	— — — —	No characteristic lesions
145	1–11	Mandler 7	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	6 to 7	+ + + —	Vesicles and pustules 6th to 7th day. Ruptured 12th day. Scab fell off by 24th day
146	1–11	Mandler 8	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	Negative	— — — —	No characteristic lesions

*Control organism in all experiments was *Staphylococcus pyogenes albus*.

TABLE VI—*Experiments with Mandler and Chamberland filtrates*

LAMB	DATE (1929)	TYPE OF FILTER*	POINT OF INOCULATION	DILUTION	PERIOD OF INCUBATION (DAYS)	RE-ACTION	REMARKS
19	1–11	Mandler 7	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	5 to 6	+ + + —	Vesicles and pustules 5th to 6th day. Ruptured 10 to 11th day. Scab fell off by 23rd day
4	1–11	Chamberland L 2	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–50,000 1–100,000	6 to 7	+ + + —	Vesicles and pustules 6th to 7th day. Ruptured 12th to 13th day. Scab fell off by 24th day
6 (Control)	1–11	Mandler 7 Chamberland L 2	Right side of thigh Left side of thigh	1–1,000 1–10,000 1–1,000 1–10,000	Negative	— — — —	This animal had previously passed through a natural attack of the disease

*Control organism in all experiments was *Staphylococcus pyogenes albus*.

can draw any definite conclusions as to whether or not a lasting immunity against reinfection is certain. Thus far, we can say that the susceptible animals that have been inoculated with the virus during this experiment have remained immune for months, also animals that have had a natural attack of the disease have also remained immune.

One should be conservative in using this virus as a prophylactic agent, because this disease as seen in this country at present is mild; to date, with over 1500 cases recorded, we have had no fatalities, and all animals made good recoveries. (See table VII.)

FIG. 5. Vesicles and pustules developing the sixth day, on the line of scarification. This animal (19) was inoculated with the virus passed through a Mandler filter 7.

SUMMARY AND CONCLUSIONS

It has been possible to demonstrate the presence of a filtrable virus in both the vesico-papular lesions and the scabs of infectious pustular dermatitis of sheep and goats.

Sheep and goats appear to be the only animals susceptible to this virus.

We were unable to obtain an infective filtrate each time with the same type of filter, thus showing that the filtrability of the etiological agent is carried out with the greatest difficulty.

TABLE VII—*Experiments with sheep that had previously passed through a natural attack of the disease*

ANIMAL	DATE (1929)	VIRUS	DILUTION	POINT OF INOCULATION	PERIOD OF INCUBATION (DAYS)	RE-ACTION	REMARKS
Ewe 2	2–4	25% glycerin	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 60 days previous to this inoculation
Wether 9	2–4	25% glycerin KH_2PO_4 (pH 7.5)	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 180 days previous to this inoculation
Wether 6	2–4	25% glycerin	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 180 days previous to this inoculation
Lamb 32	2–4	Passed through Chamberland L 2	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 60 days previous to this inoculation
Lamb 33	2–4	Phys. NaCl solution	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 60 days previous to this inoculation
Lamb 34	2–4	Passed through Mandler 7	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 30 days previous to this inoculation

TABLE VII—*Experiments with sheep that had previously passed through a natural attack of the disease—Continued*

ANIMAL	DATE (1929)	VIRUS	DILUTION	POINT OF INOCULATION	PERIOD OF INCUBATION (DAYS)	RE-ACTION	REMARKS
Ewe 148	2—4	50% glycerin	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	—	— — — —	Animal had passed through an attack of the disease 30 days previous to this inoculation
Lamb 36 (Control)	2—4	Passed through Chamberland L 2	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	6 to 7	+ + + —	Vesicles and pustules 6th to 7th day. Ruptured 13th day. Scab fell off 24th day
Ewe 11 (Control)	2—4	Passed through Mandler 7	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	7 to 8	+ + + —	Vesicles and pustules 7th to 8th day. Ruptured 13th day. Scab fell off 24th day
Ewe 84 (Control)	2—4	25% glycerin	1–1,000 1–10,000 1–50,000 1–100,000	Right side Left side	5 to 6	+ + — —	Vesicles and pustules 5th to 6th day. Ruptured 12th day. Scab fell off 23rd day

Animals that have recovered from an experimental inoculation, and those animals that have passed through an attack of the disease possess a high degree of immunity.

We were unable to demonstrate *Bacillus necrophorus* by rabbit inoculation, thus indicating that we were dealing with a different etiological factor than that causing so called lip and leg ulceration.

Infectious pustular dermatitis may assume enzootic proportions, and the outlook may appear very grave at first, but, if the animals are allowed to go untreated, recovery soon takes place.

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U. S. Civil Service Examination

The United States Civil Service Commission announces an open competitive examination for junior veterinarian to fill vacancies in the Bureau of Animal Industry, Department of Agriculture, for duty in the field. The entrance salary for junior veterinarian is $2,000 a year. Higher-salaried positions are filled through promotion.

Competitors will be rated on theory and practice of veterinary medicine and on veterinary anatomy, physiology and pathology, and meat inspection. The duties are to make antemortem and postmortem inspections of food animals and inspection of food products; the administration of tests for disease; control and eradication of disease; and sanitary inspection of establishments and plants, and related duties as directed.

Applications for this examination must be on file with the Civil Service Commission at Washington, D. C., not later than December 31, 1929.

Full information may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the United States Civil Service Board of Examiners at the post office or customhouse in any city.

A COMPARISON OF THE RAPID AND SLOW AGGLUTINATION METHODS FOR THE DIAGNOSIS OF BANG ABORTION DISEASE IN CATTLE

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The usual method of examining bovine sera for agglutinins for *Brucella abortus* is by the time-honored slow technic which involves incubation of the serum-dilution-antigen mixture for 36 to 48 hours. At the end of this time the results are read in terms of the degree of clearing in the tubes. This clearing or agglutination is in turn interpreted as indicating the concentration of specific antibodies in the serum and an animal is considered a reactor, or not, depending on the observed result in dilutions of 1:50 or 1:100 of the serum. This method of diagnosis has been practiced for a decade or more and its reliability thoroughly established.

Comparatively recently Huddleson[1,2] has proposed a new or rapid method of agglutination that eliminates the long incubation period and permits of an almost immediate diagnosis. The technic employed brings the undiluted serum and *Brucella abortus* antigen together on a glass plate where they are mixed with a match-stick or tooth-pick, slightly warmed and allowed to stand a few moments. The result is read in not more than five minutes.

Obviously, such a method has advantages that would at once recommend it to those in diagnostic laboratories, where numerous sera are examined daily, if experience demonstrated that the results were identical with those obtained by the slow technic or if the discrepancy was so slight as to be negligible.

A comparison of the results obtained by the two methods has been made by Lienhardt and Kitselman,[3] in a limited number of cases. In their study the total number of samples diagnosed as positive by the two methods was identical, although in certain series one or two more were called positive by one method than by the other. In their paper the dilution taken as being diagnostic is not stated, however.

Received for publication, May 23, 1929.

In view of the facts stated above, it was thought that a statement of the results of parallel tests on bovine sera as obtained in this laboratory might be of some interest. These results are taken from a large series as being merely representative and as pointing to certain conclusions and do not represent the total number of tests in which examination by both methods was employed. In table I, the results of parallel tests on the same sera, employing both methods of testing, are presented. For comparison the record in both the 1:50 and 1:100 dilution of the sera is included.

TABLE I—*Comparison of rapid and slow agglutination tests employing sera presumably positive*

Samples Positive in 1:50 Dilution		Samples Negative in 1:50 Dilution		Samples Positive in 1:100 Dilution		Samples Negative in 1:100 Dilution	
Rapid	Slow	Rapid	Slow	Rapid	Slow	Rapid	Slow
27	27	15	15	25	25	23	23
18	18	0	1	14	12	1	3

From these tests it would appear that when the 1:50 dilution of serum is taken as diagnostic, the results by both methods are exactly the same. However, if 1:100 is the diagnostic dilution in certain series, at least, it seems that more positives are obtained by the rapid than by the slow method. The truth of this observation becomes more obvious from an inspection of table II.

TABLE II—*Comparison of rapid and slow agglutination tests, employing sera many of which were known to be negative*

Samples Positive in 1:50 Dilution		Samples Negative in 1:50 Dilution		Samples Positive in 1:100 Dilution		Samples Negative in 1:100 Dilution	
Rapid	Slow	Rapid	Slow	Rapid	Slow	Rapid	Slow
43	41	102	104	34	27	136	143
33	27	85	91	21	21	104	104

Such results as these indicate that with some bloods, at least, more positives are detected by the rapid test than by the slow method, no matter whether the diagnostic dilution is 1:50 or 1-100.

Discrepancies such as those indicated in table II are further emphasized in table III, in which there is fairly close agreement between the total numbers of positives and negatives obtained

by both methods of testing, but in which numerous samples are diagnosed as "suspicious" by the rapid method but are "negative" by the slow technic.

TABLE III—*Showing the greater sensitiveness of the rapid test*

SAMPLES POSITIVE 1:100		SAMPLES NEGATIVE 1:100		SAMPLES SUSPICIOUS 1:100	
RAPID	SLOW	RAPID	SLOW	RAPID	SLOW
32	29	150	153	5	2
25	25	23	23	6	3
34	27	136	143	8	3
38	34	95	99	9	3

Results such as these simply serve to stress the point that the rapid method not only detects more positives than are found by the slow technic but directs attention to certain others as suspicious that would be diagnosed as definitely negative by the latter method. Further emphasis of this point is obtained by inspection of table IV.

TABLE IV—*Evidence of the sensitivity of the rapid method*

SAMPLES NEGATIVE 1:50		SAMPLES SUSPICIOUS 1:50		SAMPLES NEGATIVE 1:100		SAMPLES SUSPICIOUS 1:100	
RAPID	SLOW	RAPID	SLOW	RAPID	SLOW	RAPID	SLOW
596	731	125	63	834	884	65	27

CONCLUSIONS

From a large series of tests in which the slow and rapid methods of testing blood sera for agglutinins for *Brucella abortus* in parallel were employed it seems that:

1. The rapid method is more sensitive than the slow.
2. Numerous samples will be diagnosed positive by the rapid method that are, at most, only suspicious by the slow technic.
3. Many samples will be diagnosed as suspicious by the rapid method that are negative otherwise.
4. The rapid method will detect more quickly certain animals that are positive or should be regarded with suspicion than will the slow agglutination test.

5. Contradictory results are obtained by the two tests in a negligible number of cases.

6. The delicacy of the rapid method, together with the care necessary in preparation and standardization of the antigen used, makes it a test applicable only in the hands of experienced laboratory workers.

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Tail Waggers' Club

The Tail Waggers' Club is the name of an organization sponsored by the World League for Dog Welfare. American headquarters have been opened at 307 Fifth Ave., New York, N. Y. The World League for Dog Welfare is a non-profit organization for the promotion of dog welfare and conducts educational and philanthropic activities throughout the entire world. The President of the League is The R. Hon. The Earl of Chesterfield and the Chairman is a well-known veterinarian in the person of Prof. F. T. G. Hobday, C. M. G., F. R. C. V. S., F. R. S. E.

According to an announcement just made public, the Tail Waggers' Club was founded "for the express purpose of doing a humane service to man's best friend—the dog. It will render a service to each individual dog-owner whose pet is a member. Being organized entirely for the benefit of its canine members, all funds accumulating from membership fees are to be used for the dogs themselves. Member dogs will wear an identifying medallion, which will be registered at a central bureau, no matter where the dog resides.

One of the main functions of the Club will be to provide a means for tracing or tracking down lost or stolen members. Membership in the Club is not restricted to pedigreed dogs. On the other hand, the League is most democratic and welcomes all canines regardless of ancestry. Mutts will enjoy the same membership status as even such a well-known dog as Cora, the pet Cairn of the Prince of Wales, or the Italian Greyhound belonging to Queen Maud of Norway. Mrs. Daisy Miller has been appointed Executive Secretary of the American Branch of the League.

THE CONSTANCY OF THE AGGLUTINATION TEST IN THE DETECTION OF BACILLARY WHITE DIARRHEA*

By P. R. EDWARDS *and* F. E. HULL, *Lexington, Ky.*

Department of Animal Pathology
Kentucky Agricultural Experiment Station

Several investigators have noted that sera of hens infected with bacillary white diarrhea at times fail to agglutinate *Salmonella pullorum*. As early as 1916, Horton[1] reported that a hen which had previously been positive to the agglutination test gave negative reactions. Erickson[2] tested fifteen hens for a period of twelve months. During this time, he observed fluctuations in the reactions, some of the birds varying from positive to negative. Gwatkin[3] observed the reactions of eleven positive birds for seven months. With one exception, the results were consistent. Doyle[4] performed monthly tests on fourteen reacting hens. The tests covered a period of eleven months. During this time, three of the birds ceased to react. Beach, Halpin and Lampman[5] conducted repeated tests on 64 hens. Of these, 44 were reactors at the beginning of the experiment. The birds were tested eleven times in thirteen months. Of the 44 reactors, 18 gave at least one negative test during the course of the experiment. Some of the birds gave as many as six or seven negative tests. Such results caused the authors to recommend disposing of infected flocks instead of endeavoring to eliminate infected individuals.

Beach,[6] at the California Station, tested 70 reacting pullets at monthly intervals for twelve months. While the birds were under observation, 40 gave one or more negative tests. The number of negative tests on individual birds varied from one to eleven. In some instances the birds gave alternate positive and negative tests. *S. pullorum* was isolated from birds that had given three consecutive negative tests before death. *S. pullorum* was isolated also from the ovaries of birds which had never yielded a positive agglutination test. Kaupp and Dearstyne[7] tested 29 birds at monthly intervals for fourteen months. During this period, three of the hens ceased to react. The same workers tested 71 birds for a period of six months. During this period three birds gave negative reactions. They were able to

The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director. Received for publication, July 22, 1929.

show also that these birds laid infected eggs during the period their agglutination reaction was negative. In spite of the fluctuations observed in the tests, 90 per cent of the birds tested fourteen months and 98 per cent of the birds tested six months would have reacted at any time during the course of the investigation.

Newsom, Cross and Ufford[8] performed repeated tests on 59 birds. The length of time the birds were kept under observation varied from 12 to 24 months. The number of tests upon individual birds varied from 4 to 21. Under these conditions 13 of the birds were consistently negative, 25 were consistently positive, and 21 gave variable reactions. The writers concluded that the agglutination test should be applied to flocks at frequent intervals to remove all infected birds.

The results cited above indicate that the agglutination test for bacillary white diarrhea may not be such an accurate method of diagnosis as has long been supposed. The results obtained by Beach, Halpin and Lampman[5] and Beach[6] are particularly discouraging to persons interested in the control and eradication of bacillary white diarrhea through the application of the agglutination test.

Experimental Work

In an effort to determine the amount of variation in the agglutination test as it is conducted in this laboratory, 93 hens which had reacted to the agglutination test were collected and placed under observation. These birds were not a selected group. They were made up of reacting birds removed from farm flocks as infected individuals. The hens were placed in a house 30 feet by 15 feet and allowed to range over a large yard at all times. They were tested at monthly intervals for one year.

The antigen used in the test was composed of three strains of *S. pullorum* which were known to be agglutinable. The organisms were suspended in carbolized saline solution containing 0.85 per cent NaCl and 0.5 per cent phenol. The turbidity of the antigen was 0.5 on the McFarland scale. Duplicate tests were run using an antigen to which had been added 1 per cent N/1 NaOH just previous to use. Two dilutions were employed in the tests: 1 to 40 and 1 to 80. Partial agglutination in either dilution was considered indicative of infection.

The tests which have been performed on these birds have been surprisingly consistent. During the period of observation, 984 tests were made on the 93 hens. Of these 984 tests, only 6

were negative reactions. These negative tests were confined to 4 birds. The records of the tests of these 4 birds are given in table I. The bird H197 (table I) is the only bird giving well defined (complete or nearly complete) agglutination at 1-40 at the beginning of the experiment that gave a negative test. Of 874 tests performed on such birds, only one negative test was obtained. The remaining five negative tests were confined to birds giving only a weak partial agglutination at 1-40 at the beginning of the experiment.

No attempt was made to determine the titers of the sera of the birds being tested. However, using only the two dilutions mentioned above, variations in the titers of the individual hens were apparent. At times the titer of serum which caused com-

TABLE I—*Agglutination reactions and autopsy findings in four birds*

BIRD	DATE OF TEST												
	4–25–28	5–24–28	6–25–28	7–25–28	8–22–28	9–25–28	10–24–28	11–26–28	12–18–28	1–23–29	2–26–29	3–25–29	AUTOPSY
H197	++	++	++	++	+—	±—	— —	±—	++	±—	±—	±—	—
165	± ±	±—	± ±	± ±	± ±	— —	± ±	— —	— —	±—	±—	± ±	—
H462	±—	± ±	± ±	± ±	±—	±—	± ±	±—	±—	— —	±—	±—	+
H107	± ±	± ±	± ±	± ±	— —	±—	± ±	±—	++	± ±	± ±	± ±	+

± — = partial agglutination at 1-40 no agglutination at 1-80.
± ± = partial agglutination at 1-40 and 1-80.
++ = complete or almost complete agglutination at 1-40 and 1-80.
— — = no agglutination at 1-40 or 1-80.

Autopsy:
— = *S. pullorum* not recovered.
+ = *S. pullorum* recovered.

plete agglutination at 1 to 80 would drop until only a partial agglutination at 1–40 would be observed. The titers of the sera of other birds increased during the period. However, it was only in the cases of the four birds previously noted that the titer became so low that no agglutination was present at 1-40.

During the course of the experiment 5 birds were lost. Post-mortem examinations were made of the remaining 88 hens. *S. pullorum* was recovered from 80 hens, *E. sanguinaria* from 1, streptococci from 1 and in 6 cases the examination was negative.

In the case of the hen from which *E. sanguinaria* was isolated the organism was apparently confined to the ovary. The organisms were not isolated from the heart, spleen or liver. The ova were discolored and misshapen, resembling ova infected with

S. pullorum. This hen was in apparently good health, being killed for postmortem examination at the end of the experiment. No other cases of fowl typhoid occurred in the flock.

SUMMARY

While variations have been observed in repeated agglutination tests upon infected birds, they have been slight. The variations noted in this experiment have not been so extensive as those noted by some workers. In 984 tests upon infected individuals, only six negative tests were obtained. Five of these six negative reactions occurred among hens which, at the beginning of the experiment, gave weak, partial reactions to the test.

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Mule and Farmer

SOME TAX PUZZLES

If a young man inherits a fortune and spends his time getting a nice coat of tan at Palm Beach or losing money at Monte Carlo, the government takes a certain part of his income.

If a doctor spends his days working hard and his nights answering emergency calls, wearing himself out early, the government takes the same percentage of his income.

If a farmer driving a mule is struck by a train and pays forty dollars to a veterinary for the mule and a hundred dollars for his own doctor's bill, the government allows deduction for the mule, but none for the farmer.

That seems unreasonable.

New York State proposed to reduce by 25 per cent the income tax on *earned* incomes, as compared with a tax levied on *unearned* income.

If a man owns an oil well or a coal mine he is permitted to make a deduction from his income tax because of "depletion." There is less oil in his well, less coal in his mine at the end of a year.

There is also less power in a professional man's body, less energy in a business man's brain at the end of a year. Why shouldn't they charge for "*depletion*" also?

—Editorial in the Detroit (Mich.) *Times*.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

TULAREMIA OF ANIMALS COMMUNICATED TO MAN

By ROBERT GRAHAM *and* FRANK THORP, JR.

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University of Illinois, Urbana, Illinois

Tularemia is an infectious disease of rodents and man caused by *B. tularense*. It occurs in nature as a fatal bacteremia of

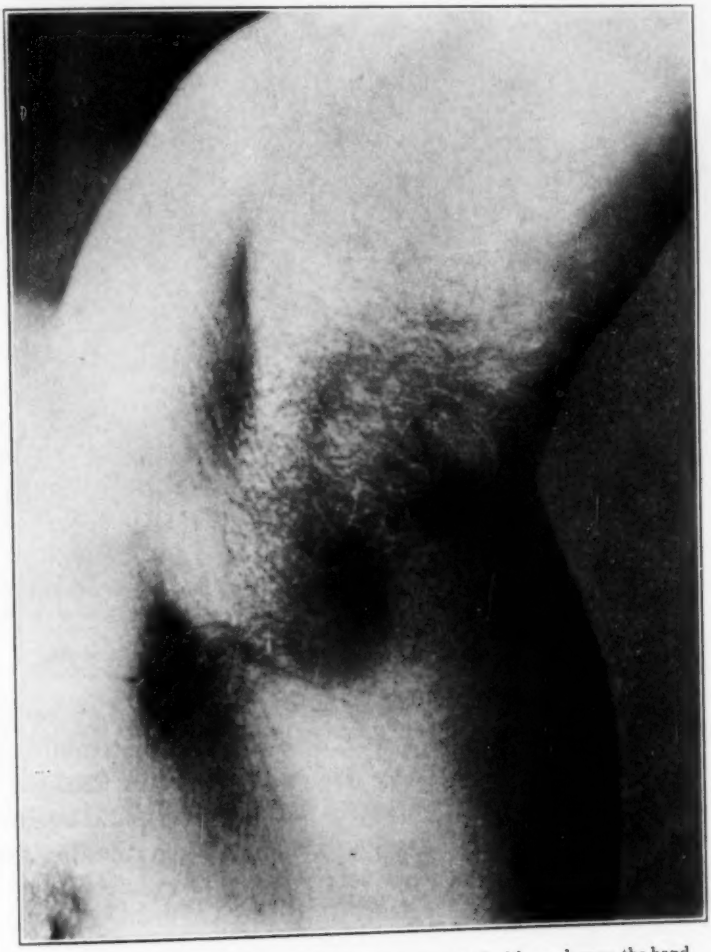

FIG. 1. The ulceroglandular type of the disease associated with an ulcer on the hand.

wild rodents, especially rabbits and hares. More recently the disease was described in sheep by R. R. Parker and James Dade.*

Secondary to the infection in animals, tularemia is a disease of man and is transmitted from rodents to man by the bite of an infected blood-sucking fly or tick, or by contamination of the hands with portions of the internal organs or with the body fluids of infected rabbits, flies or ticks. The disease has been reported in nineteen states, and was first encountered in ground squirrels in California, by McCoy, in 1911.

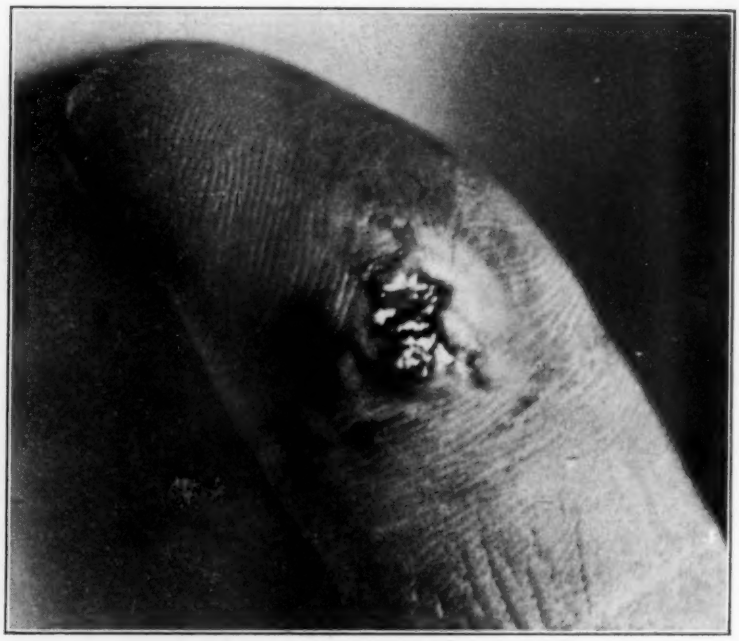

FIG. 2. Tularemia ulcer on thumb following hunting and handling of rabbits.

During the past few years it has been reported in certain localities of Illinois. Last winter a patient suffering from tularemia-like ulcers came under the care of Doctor Christie, of Champaign, Illinois. The lesions on the hands and regional lymph-glands were suggestive of tularemia. Agglutination tests of serum from two patients conducted at the Laboratory of Animal Pathology and Hygiene, University of Illinois, confirmed the diagnosis.

*Parker, R. R., and Dade, James: Tularemia: Its transmission to sheep by wood tick, *Dermacentor andersoni* Stiles. JOUR. A. V. M. A., lxxv (1929), n. s. 28 (2), pp. 173-191.

Tularemia may assume four different types in man: (1) the ulceroglandular type; (2) the oculoglandular type, with an involvement of the conjunctiva; (3) the glandular type, without primary lesions; and (4) the typhoidal type, without primary lesions and without glandular enlargements. The accompanying photographs illustrate the ulceroglandular type.

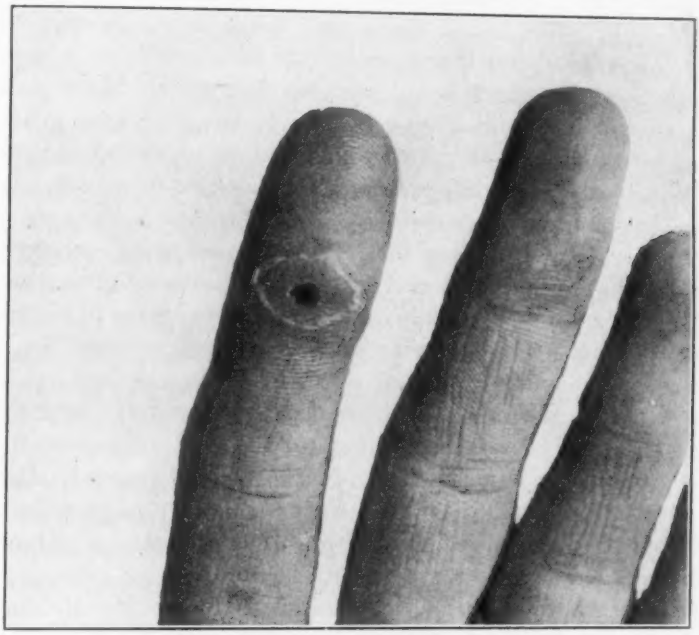

FIG. 3. One of the rabbits was given to a neighbor and in 10 days a malignant ulcer developed on the index finger. The photograph was taken several weeks after initial ulcer.

Veterinarians, through a knowledge of the gross pathological lesions of tularemia in human beings and the epizoological significance of the disease in animals to man, have, through suggestions, been helpful to physicians in diagnosing the disease in man.

HEMORRHAGIC SEPTICEMIA IN A MINK

By HENRY V. LEWIS, *Brookings, S. Dak.*

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Increased interest in fur-farming has created problems in diseases of fur-bearing animals raised in captivity. This in turn has presented many new problems to veterinarians for

solution. When interest in fur-farming first began, there was no literature available on diseases of fur-bearing animals. Such literature is now accumulating, but it is far from being as complete as the literature that deals with diseases of domestic animals. The following case report is presented with the hope that it will be of value to fellow veterinarians who may be called to investigate diseases of mink.

History: This report deals with mink raised in captivity. The owner has some twenty mink that he is raising on a special mink food, prepared by a company located in Minneapolis. This company recommends a feed of chicken once a week. The owner has been following these instructions by giving the mink a feed of heads and internal organs of chickens once each week. He obtains these by-products at a local produce house. As per schedule, on the evening of October 3, each mink received a feed of the heads and internal organs of chickens. On the evening of October 4, one male mink was showing evidence of sickness.

Symptoms: The following symptoms were reported by the owner: The animal was very restless, doubling its body as if in great pain. It seemed hard for it to breathe and slight hemorrhage was noticed coming from the nostrils. Occasionally it acted as if there might be some obstruction of the throat. Death occurred very suddenly. The owner was unable to do anything although he was able to pick up the animal without being bitten. This animal was presented at the laboratory for autopsy, on the morning of October 5, after being skinned by Mr. Heim.

Lesions: Autopsy of the mink carcass revealed hemorrhage into the nasal passages and trachea. The lungs were typical of hemorrhagic septicemia in other animals. The surface of the heart was dotted with petechiae. A slight catarrhal enteritis was present throughout.

Laboratory examination: The heart was seared with a red-hot spatula and blood smears made from the heart-blood. These blood smears were stained with both Gram's and Wright's stains. In both cases, organisms of a bipolar nature were present.

Cultures inoculated: Veal-agar plates were made and streaked with a loopful of blood from the heart. Dextrose broth medium was made neutral and sterilized in fermentation tubes. After cooling, the fermentation tubes were inoculated with a loopful of the heart-blood. These cultures were incubated for forty-eight hours at 37° C. The veal-agar plates showed many shiny colonies about the size of a pin-head. Smears were made and stained,

revealing bipolar organisms. The dextrose broth medium showed no gas production and very slight acid formation. Bipolar organisms were isolated from this medium.

Animals inoculated: Two mice each received one loopful of heart-blood subcutaneously. They were found dead twenty hours after injection. Spleen smears stained by Wright's method revealed many short bipolar rods. A healthy normal rabbit received 0.1 cc of heart-blood intravenously. It was found dead twelve hours after intravenous injection. Autopsy revealed enteritis, pneumonia, and petechiae on the heart. Laboratory examination of the heart-blood revealed the presence of large numbers of bipolar organisms. Pieces of the liver and kidneys of this rabbit were sectioned and stained, disclosing the fact that the microorganisms were present in great number throughout the kidney and liver tissue.

October 9, a healthy normal chicken received 0.1 cc of the heart-blood of the rabbit, injected intramuscularly in the breast muscle. The first symptom in the chicken was refusal to eat after about ten hours. A severe diarrhea developed soon afterward. The bird was found dead thirty-six hours after injection. Autopsy revealed petechiae on the heart and serous membranes. Laboratory examination of the heart-blood showed numerous bipolar organisms resembling those of fowl cholera.

Conclusions: Further work is needed before very definite conconclusions may be drawn. Inability to secure mink for experimental purposes has necessitated stopping at this point. However, it can be safely said this disease was either a primary septicemia of mink or septicemia contracted from feeding offal from cholera poultry. The owner has ceased feeding poultry by-products and reported that he has had no losses since.

In order to form definite conclusions, it would be necessary to feed mink the carcasses of poultry that died from cholera.

A LIVER TUMOR IN A LION

By L. P. Doyle, *Lafayette, Ind.*

Department of Veterinary Science
Purdue University Agricultural Experiment Station

An aged male lion died after a series of convulsions extending over four days. The convulsions occurred at intervals of five to six hours. Each attack was followed by a short period of lethargy, after which the animal became apparently normal. Previously

the lion had not been seen to show symptoms of any illness. Two or three days before the onset of symptoms, the animal was given a dose of "worm medicine."

Autopsy showed a well-nourished carcass, free from gross lesions except a large tumor in the liver and a well-marked congestion of a portion of the small intestine. The tumor was located in the right central lobe of the liver, near the ventral border. It was grayish in color and had an irregularly lobulated surface. The whole mass was surrounded by a fibrous capsule. The consistency of the tumor was quite firm except in areas where necrosis had occurred. The necrotic areas were friable but free from liquefaction. The neoplasm was roughly circular in outline,

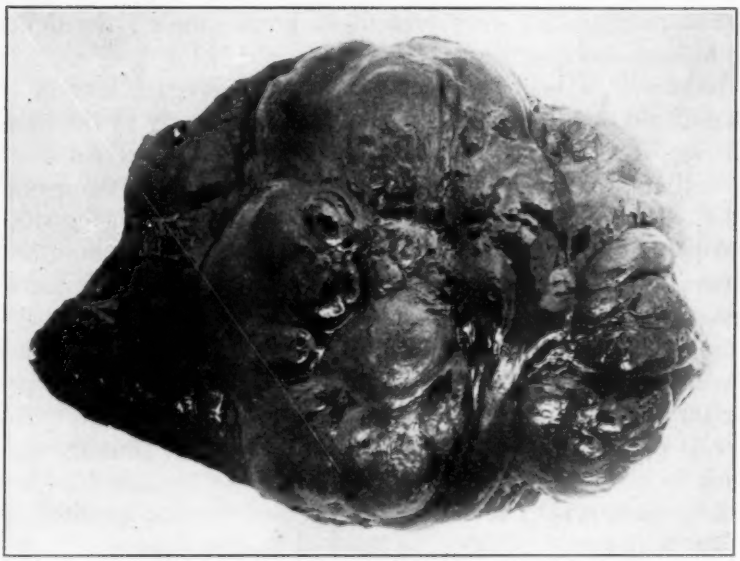


FIG. 1. A photograph showing the gross appearance of the tumor. The tumor is circular in outline. The projecting portion is part of the lobe of the liver.

20 centimeters in diameter and 8 centimeters thick. It weighed 1,900 grams.

Microscopic examination of the tumor showed that it was made up largely of generally roundish cells, closely resembling liver cells. A few of the tumor cells were of great size and had large, single, hyperchromatic nuclei. The cytoplasm of nearly all the cells contained clear vacuoles which were probably fat spaces. The arrangement of the cells was generally not definite. In some areas, however, there was a perithelial arrangement and in others

an alveolar structure. Very few of the cells had a definite cord-like arrangement; and the majority of these were definitely recognizable as old hepatic cords which had survived the invasion of the liver by the tumor. There were a good many blood-vessels present, with well-developed walls; and also many large blood-spaces with very thin walls. Rather wide bands of connective tissue passed through the tumor, but there was very little finer stroma between the new-formed cells. It appears justifiable to regard this neoplasm as a true hepatoma or primary tumor of

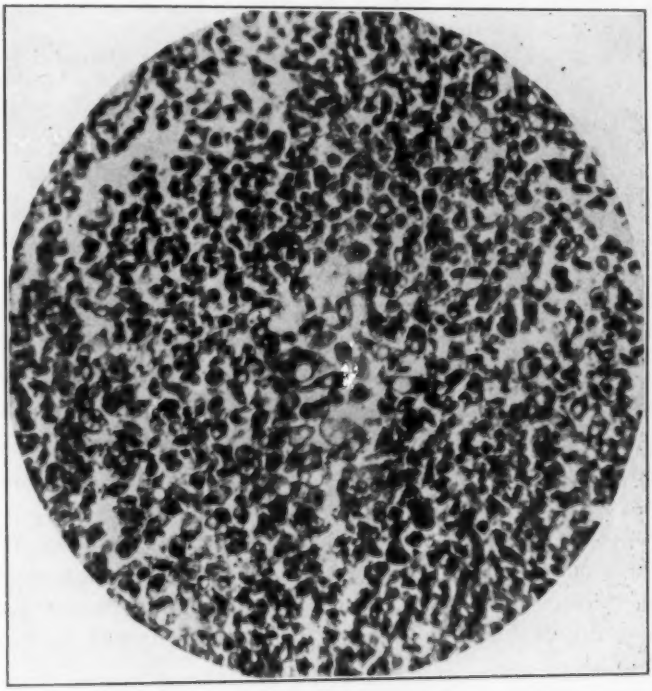


FIG. 2. A photomicrograph showing type of cell in the tumor.

the liver which originated from the parenchymatous liver cells rather than from the bile-duct epithelium.

It is not certain, of course, that there was any causal relation between the tumor and the symptoms which the lion showed. The tumor may have interfered in a mechanical way with the functioning of the intestine; or it may have reflexly induced symptoms. Moreover, absorption from the necrotic portion of the tumor may have caused trouble. The administration of the "worm medicine" can not be considered as free from possible

responsibility for the symptoms. Veterinarians have frequently observed that marked convulsive symptoms in other species of felines, as well as canines, often follow the giving of certain alleged worm remedies.

Darwinism

A hungry toad and a June bug bright
Happened to meet on a summer's night.
Now the toad knew the bug was bitter and tough.
He was sure he could never digest that stuff,
But he had to eat, so he took a chance
And he swallowed the bug with a sidelong glance
Which revealed, too late, a great big snake
Craftily following in his wake.
Just a sudden dart of that glistening head
And quicker than scat, the snake was fed.
For he swallowed the toad with the bug inside
And crawled in the rocks to sleep and hide.

M. L. PLUMER.

Large Area Released from Tick Quarantine

Notice of the release of 10,358 square miles of additional territory, in four southern states, from federal quarantine on account of cattle ticks, is contained in Bureau of Animal Industry Order No. 321 just issued by the U. S. Department of Agriculture, effective December 1.

The order releases the following counties from the quarantine: Clarke County in Alabama; Baker, Columbia, Suwannee and Union counties in Florida; Amite, Clarke, Jasper, Jefferson Davis, Lawrence and Simpson counties in Mississippi; and Bell, Red River, Upshur and Wood counties in Texas.

The order also requarantines Jefferson Davis Parish, Louisiana, the area of which is 729 square miles; and it continues the existing quarantine in the state of Arkansas and the territory of Porto Rico.

With the release of Clarke County, Alabama, all of Alabama becomes free from the tick embargo, thus making Alabama the tenth state to join the list of states which, through tick eradication, have worked their way above the tick-fever quarantine line. The states which previously had reached this goal are California, Georgia, Kentucky, Missouri, North Carolina, Oklahoma, South Carolina, Tennessee and Virginia.

REVIEWS

CANINE NURSING. David Eric Wilkinson, M. R. C. V. S. 40 pages. 6 figures. Watmoughs, London, 1929. 3sh 6d ($.90).

In the preface the author states frankly that this little book has been prepared at the request of a number of dog-owners and breeders and that the subject is being presented in a non-technical way for the benefit of lay readers. Chapter I is introductory. Chapter II describes the equipment of a well-appointed canine hospital. Chapter III discusses surgical nursing, including the dressing of wounds, bandaging, etc. Chapter IV covers restraint for surgical operations, as well as devices for preventing dogs from disturbing dressings or self-inflicting injuries. Considerable space is given to describing the Elizabethan collar. Chapter V presents the subject of medical nursing, including the administration of medicines. Chapter VI is devoted to the nursing of patients affected with brain and nervous disorders. Chapter VII is given over to dietetics, which is discussed quite fully. The author advises against the use of alcoholic stimulants in canine practice. Chapter VIII concludes the book and contains a discussion of general nursing. Veterinarians will undoubtedly get more than a few good suggestions by carefully perusing this little book.

SUMMARIZED PROCEEDINGS OF THE AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE, 1925-1929. Edited by Burton E. Livingston *et al.* Washington, D. C., 1929. Part I, 185 pages. Part II, 1,006 pages.

This report covers the meetings of the A. A. A. S. held in Boulder, Colo. (1925); Portland, Ore. (1925); Kansas City, Mo. (1925); Philadelphia, Pa. (1926); Nashville, Tenn. (1927); and New York, N. Y. (1928), and includes an account of the organization and work of the Association (Part I). The larger portion of the book is taken up with a list of the sustaining members, life members, and emeritus life members, and a directory of the fellows and other members (Part II). The two parts are separately paged. The publication of a report of this kind represents a tremendous amount of detailed work, all of which appears to have been done with an unusual amount of care.

TREATMENT OF CANINE DISTEMPER WITH THE POTENTIZED VIRUS. Horace B. F. Jervis, V. S. 40 pages. Ehrhart & Karl, Chicago, 1929.

An urgent plea for veterinarians to employ homeopathy and homeopathic principles in the treatment of canine distemper. Potentized virus is the virus of canine distemper potentized as per homeopathic methods. According to the author, immunity conferred by this agent is direct. It is administered orally and the author contends that by reason of the fact that certain infectious diseases gain entrance to the body via the alimentary canal, immunizing agents entering the body the same way should produce immunity. The first part of the book is a recital of the author's failures with medicinal agents and the various biological products advocated for the prevention and treatment of distemper. Then follows an account of the author's experiences with potentized virus, both as a curative and as a prophylactic.

HIGIENE MODERNA DE LA LECHE (Modern Milk Hygiene). Prof. Dr. Kurt Schern, Director of Milk Hygiene Service, Municipality of Montevideo, Uruguay. 127 pages with 17 figures. Garcia Morales, Montevideo, 1929.

The bulletin, dealing with the public milk supply of Uruguay, is divided into four chapters. Chapter I. A New View of Milk Hygiene Based upon Bacteriological Investigation. Chapter II deals with "Vitamins and Their Relation to Milk Hygiene." Chapter III. Modern Legislative Control of Milk Hygiene. Chapter IV. The Education of the Producer and Dealer by Technical Instruction in Hygiene, Economics and Legal Control.

Tables are presented showing the results of examinations of samples of milk obtained in Montevideo during a period of two years. Of the samples examined, 101 showed 500,000 bacteria per cc, 814 samples showed a bacterial count ranging from 500,000 to 4,000,000 bacteria per cc, 735 samples ranged from 4,000,000 to 20,000,000 bacteria per cc and 283 samples showed more than 20,000,000 bacteriae per cc.

The bulletin very clearly demonstrates the necessity of clean milk and also shows the various methods that should be followed to obtain a safe milk supply.

N. S. M.

ABSTRACTS

A Contribution to the Knowledge of Pyaemic Forms of Infection in Sheep. Hilding Magnusson. Jour. Comp. Path. & Ther., xlii (1929), 2, 73.

A pyemic disease, localized in the subcutaneous soft parts of the head and the associated lymph-glands, has been encountered in four different flocks of sheep. Abscesses have been found also in some cases in the lungs, the liver, the kidneys, and the udder. The abscesses contained a plastic, viscous pus, in which were present small numbers of an organism identical with *B. purifaciens* Christiansen. Blood serum of infected sheep agglutinates the organism in dilutions of 1 to 1280. It is pathogenic for white mice inoculated intraperitoneally and for goats and horses injected subcutaneously. It is pyogenic for sheep when injected subcutaneously, intravenously, intraperitoneally and intramammarilly.

Cultural Studies of an International Collection of Clostridium Botulinum and Parabotulinum. J. B. Gunnison and K. F. Meyer. Jour. Inf. Dis., xlv (1929), 2, p. 119.

Cultural, biochemic and serologic studies, conducted with the simplest medium and by the simplest technic, with 53 strains concerned in human and animal botulism, have shown that the action on native protein, the peptolytic property, the fermentation reaction, the agglutination and toxin-antitoxin neutralization tests are of importance for identification and classification of the various strains. The authors observe that the identification and classification of organisms involved in human and animal botulism become more complex and difficult as new strains are added to the list of already known types.

The Cytology and Microchemistry of Mycobacterium Tuberculosis. Georges Knaysi. Jour. Inf. Dis., xlv (1929), 1, p. 13.

The young cell of *Mycobacterium tuberculosis* consists of a membrane presenting thickened areas and granular appendages on its internal surface which surrounds a very dense, deeply staining cytoplasm permeated by a vacuolar system and in-

closing dense round or oval hyperchromatic granules. The author was unable to substantiate the claims of various investigators of the existence of a wax or fat sheath around or in the cell of the tubercle bacillus. He suggests that the presence of such substances as waxes, fats and various fatty acids and higher alcohols in cultures of this organism probably arises through degenerative processes in the structure of the cell and that these actually start long before the cell is dead.

UNDULANT FEVER. A. V. Hardy. Jour. Amer. Med. Asso., xciii (1929), 12, p. 891.

The author gives a very thorough review of this disease and calls attention to the fact that it is not a disease of the future but one which demands the immediate attention of the medical world. He observes that the pathologic lesions and clinical signs of *Br. melitensis* infections in animals show a definite correlation to those existing in the human. The epidemiologic data, based on the reports of more than a thousand cases of undulant fever in the United States, indicate that cattle and hogs with contagious abortion are the source of these infections. He urges the cooperation of veterinarians.

THE OCCURRENCE OF BACILLUS SORDELLI IN ICTEROHEMOGLOBINURIA OF CATTLE IN NEVADA. Ivan C. Hall. Jour. Inf. Dis., xlv (1929), 2, p. 156.

Two strains of a previously unidentified anaerobe isolated in 1919 and 1921 by Dr. L. R. Vawter, of Reno, Nevada, from typical cases of icterohemoglobinuria in cattle and against which he prepared an effective antitoxic serum, have been proven to belong to the species *B. sordelli*. While this is the third time that this organism has been found to occur in the United States, it is the first time in which it has been found to be associated with cattle. From his work with this organism in rabbits and guinea pigs, the author concludes that the Nevada strains of *B. sordelli* are probably secondary invaders in icterohemoglobinuria, the primary cause of which is believed to be *Bacillus hemolyticus*.

THE SUITABILITY OF VARIOUS BACTERIA AS FOOD FOR HOOKWORM EGGS. Oliver R. McCoy. Amer. Jour. Hyg., x (1929), 1, p. 140.

All of the evidence from the experiments indicates that living bacteria constitute the essential food utilized by hookworm

larvae in developing to the infective stage. In autoclaved fecal cultures very few larvae survived after ten days and these few usually showed only slight growth. Larvae grew to the infective stage in suspensions of bacteria in normal salt solution. The larvae were able to grow on 22 of the 25 species of bacteria which were tested, but different organisms were not equally suitable for growth.

A Study of Moisture Requirements of the Eggs of the Horse, the Dog, Human and Pig Ascarids. G. F. Otto. Amer. Jour. Hyg., x (1929), 2, p. 497.

The eggs of the horse ascarid, *Parascaris equorum*, were more resistant to desiccation than those of the dog ascarid, *Toxascaris canis*, and human and pig ascarids, *Ascaris Lumbricoiaes* and *A. suum*. As the temperature rises, the moisture requirement of the egg increases. The effects of this temperature-moisture relationship probably account for the death of many eggs exposed to the sun. However, the difference in temperature of the different types of soils in the sun is apparently what makes ascaris eggs die more rapidly on one than another.

The Effect of Exercise on the Size of Normal Hearts and Enlarged Hearts of Dogs. Harold J. Stewart. Jour. Clin. Invest., vii (1929), 3, p. 339.

The author studied the effect of running on a treadmill, on the size of the hearts of dogs. A decrease in the size of the heart of normal dogs occurred following voluntary exercise. When dogs in which the heart is enlarged in consequence of artificially created valvular defects, but in which there is presumably no myocardial disease, and in which there are no signs of heart failure, are subjected to exercise, the size of the heart likewise decreases.

Animal Strains of Haemolytic Streptococci. J. Smith. Jour. Path. & Bact., xxxii (1929), 3, p. 401.

A detailed study of the cultural characteristics of a number of strains of hemolytic streptococci isolated from cows, horses, and guinea pigs has not shown a possible method of differentiating animal from human strains. The exotoxin produced by certain strains obtained from cows and horses appears to be identical with the exotoxin obtained from human strains.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Captain Thomas A. Breen, now at Station Hospital, Fort Sam Houston, Texas, has been directed to report to the president of the Army Retiring Board, at headquarters 8th Corps Area, for examination by the Board.

Veterinary Reserve Corps

New acceptances

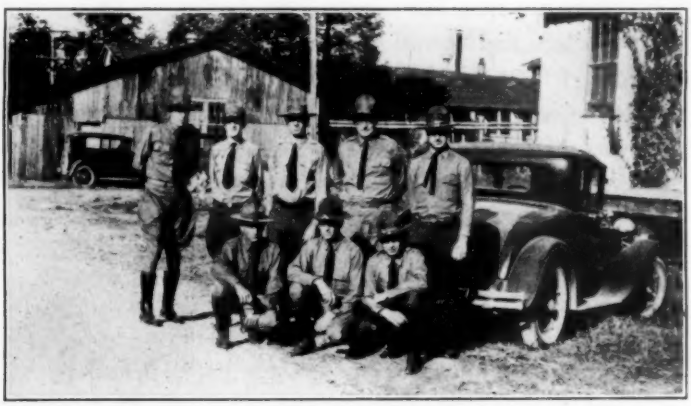
Arzberger, Walter Wm.	Captain	1120 Jones St., Watertown, Wis.
DeCamp, Clayton Earl	2nd Lt.	50 Orton Place, Buffalo, N. Y.
Ericson, Eric Oscar	Captain	5127 Dodge St., Duluth, Minn.
Isbell, George P.	Captain	314 E. 9 St., Hopkinsville, Ky.
Juen, Henry Thomas	2nd Lt.	R. R. No. 1, El Paso, Texas
O'Flaherty, Fred	1st Lt.	2858 McKinley Ave., Cincinnati, Ohio.

Promotions

Hudson, Bentley Farnell	to 1st Lt.	105 N. Main St., Moweaqua, Ill.
Kutz, Harry Cooper	" Captain	41 Elmira St., Mansfield, Pa.
Shigley, James Fremont	" Captain	118 S. Sparks St., State College, Pa.
Wetter, Charles Herbert	" Captain	Princeton, Minn.
Wright, Charles Clinton	" Major	320 Arts Bldg., Vancouver, Wash.

At Camp Knox, Kentucky

The accompanying photograph shows a group of veterinarians, all holding commissions in the Veterinary Reserve Corps, who were at Camp Knox, Ky., from August 11 to 24, 1929. All expressed themselves as being extremely well pleased with the training and experience they received during their encampment.



VETERINARIANS AT CAMP KNOX

Front row (sitting left to right): Capt. T. B. Hinkle, 1st Lt. T. J. Stearns and 2nd Lt. F. C. Hamilton.

Rear row (standing left to right): 2nd Lt. Lester C. Neer, Maj. F. C. Kneup, 2nd Lt. L. R. Sullivan, Capt. P. T. Gillie and 2nd Lt. A. A. Hiett.

MISCELLANEOUS

HOW MUCH ARE YOU WORTH ON THE HOOF?

By PHILIP P. JACOBS

Any baby boy who has potentialities of earning as much as $2,500 a year when he grows up, is worth at birth $9,333 according to an estimate made by Dr. Louis I. Dublin, statistician of the Metropolitan Life Insurance Company, after an extended study of the value of human life. Dr. Dublin's studies are told in detail in his interesting book, *Health and Wealth.*

The sum of $9,333 is the amount needed to be put at interest at 3½ per cent to bring up a child to eighteen, in order that he may be able to earn $2,500 a year. When the child is eighteen years of age, he is worth "on the hoof," as cattle buyers say, $10,000 according to Dr. Dublin. But more than that, if you capitalize his future earnings, at the age of eighteen, this boy is worth well in excess of $41,000 gross. In other words, he has the possibility of earning that amount of money.

Up until the age of eighteen, he is not a producer. He is a consumer and his parents and the community spend lavishly on him. After he reaches the age of eighteen, however, he is a producer and generally he earns for the community and for himself three times as much as he spends. Deducting from his gross future earnings his prospective future expenditures, at the age of eighteen his net earnings will be $29,000. The maximum value of a man in the $2,500 income class, is reached at the age of twenty-five, when the present worth of his net future earnings is $32,000. By the time he is fifty, his future earnings have shrunk to $17,510 and by the time he is sixty to $8,500.

Computing on this basis the value of the 60,000,000 male persons and estimating the value of women in general as one-half that of men, Dr. Dublin arrives at the conclusion that the vital or health capital of the nation is equivalent to $1,500,000,000,000. The national wealth in material assets is capitalized at $321,-000,000,000. Thus the vital wealth or the wealth that Uncle Sam has invested in the bodies and health of his citizens is worth

five times that of his mines, factories, farms, and other material goods.

But suppose tuberculosis or some other deadly disease develops in a people, what happens to this vital wealth? Over night a producer whose earning capacity is worth three times what he spends, is converted into a dead loss to himself and to the community. From an asset he is transformed into a liability. Is it any wonder that we spend millions of dollars to fight tuberculosis, a disease which annually alone costs the United States over $500,000,000?

When, therefore, anyone complains to an intelligent business man that the cost of nurses, health officers, hospitals, preventoria, and other agencies to fight tuberculosis and to care for various types of sickness are expensive and that they raise taxes, an all too common complaint, the attitude of the business man should be very clearly this: that the community as a whole can make no finer investment that that of preventing preventable diseases. Every time a young man of eighteen in the $2,500 income class is saved from tuberculosis, the community adds $29,000 net to its resources. Every time a young man of twenty-five in the $2,500 income class is saved from tuberculosis, the net amount of $32,000 is added to the community's resources. For men in the higher income classes, the saving is proportionately greater.

Every intelligent effort, therefore, that is made by such organizations as the national, state and local anti-tuberculosis associations, the health departments, tuberculosis sanatoria and other institutions to fight tuberculosis, is a move not merely toward life saving, but it is a move also in the direction of saving vast sums of money for the community.

Christmas seals are investments in health and in the business prosperity of a community. Every dollar's worth of Christmas seals that you buy adds to the health and the wealth of your community, and makes it a better place in which to live.

Montana to Study Animal Diseases

Dr. D. M. Warren (O. S. U. '24), of Urbana, Ohio, has been added to the force of the Montana Livestock Sanitary Board and will conduct the diagnostic work in the laboratory that has been under the supervision of Dr. Hadleigh Marsh for several years. Following graduation from Ohio State University, Dr. Warren was a member of the staff of the Veterinary Department of the Wisconsin Agricultural Experiment Station for one year. From

there he went to the College of Veterinary Medicine, State College of Washington, as an instructor. For the past three years, he has been in practice at Urbana, Ohio.

The Montana Livestock Sanitary Board and the Montana Agricultural Experiment Station have entered into a cooperative agreement to carry on veterinary research at the Experiment Station, at Bozeman. This work is to be carried on under the general direction of a committee consisting of the Director of the Experiment Station, the executive officer of the Sanitary Board, the head of the Department of Veterinary Science of the State College of Agriculture, the veterinary pathologist employed jointly by the Sanitary Board and the Experiment Station, a representative of the Montana Wool-Growers' Association, and a representative of the Montana Stock Growers' Association. Acting under this general advisory committee, there is a Project Committee consisting of the executive officer of the Sanitary Board, the head of the Department of Veterinary Science of the State College of Agriculture, and the veterinary pathologist. The present personnel of the Project Committee consists of Dr. W. J. Butler, Chairman, Dr. Howard Welch and Dr. Hadleigh Marsh. The laboratory staff consists of Dr. Marsh, Dr. Welch and Dr. Erwin Jungherr. This arrangement has necessitated the employment of another veterinarian to handle the diagnostic work previously done by Dr. Marsh. Dr. Warren has taken over this work. Dr. Marsh will retain his connection with the Sanitary Board, but will have to spend most of his time at Bozeman and will make his headquarters there.

This organization was the result of a demand on the part of the live stock interests, particularly the wool-growers, for more investigation of animal diseases in Montana. The combination of the resources and contacts of the Livestock Sanitary Board with those of the Experiment Station, and the backing of the live stock organizations should enable this cooperative organization to accomplish something worth while in the field of veterinary research.

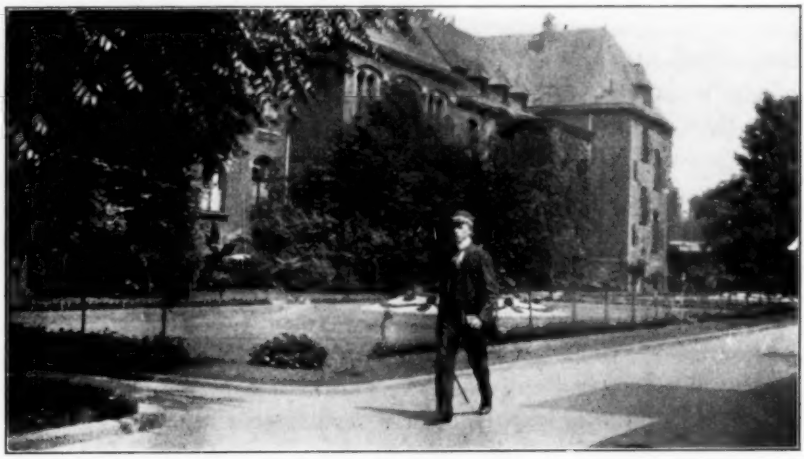
Doctor Sigler Featured

Under the caption, "Indiana Folks You'd Like to Know," the September 28, 1929, issue of *The Prairie Farmer* featured Dr. T. A. Sigler, of Greencastle, Indiana. The article included a photograph of the well-known Hoosier veterinarian and, in addition to paying Dr. Sigler a well-deserved compliment, gave the veterinary profession some nice publicity.

International Veterinary Congress

The following is the personnel of the Canadian National Committee of the International Veterinary Congress:

S. F. Tolmie, V. S., Premier of the Province of British Columbia, Victoria, B. C.
C. D. McGilvray, M. D. V., Dean, Ontario Veterinary College, Guelph, Ont.
F. T. Daubigny, D. M. V., Registrar, College of Veterinary Surgeons of the Province of Quebec, Montreal, Que.
A. A. Etienne, D. M. V., President, College of Veterinary Surgeons of the Province of Quebec, Montreal, Que.
George Hilton, V. S., Veterinary Director General, Federal Department of Agriculture, Ottawa, Ont.
E. A. Watson, V. S., Chief Pathologist, Animal Diseases Research Institute, Federal Department of Agriculture, Hull, Que.
A. E. Cameron, V. S., Chief Veterinary Inspector, Health of Animals Branch, Federal Department of Agriculture, Ottawa, Ont.
C. A. Mitchell, V. S., Pathologist, Animal Diseases Research Institute, Federal Department of Agriculture, Montreal, Que.
Arthur Gill, V. S., President, Nova Scotia Veterinary Association, Truro, N. S.
E. H. Cook, V. S., President, New Brunswick Veterinary Association, St. Stephen, N. B.
W. H. Pethick, V. S., President, Prince Edward Island Veterinary Association, Charlottetown, P. E. I.
J. S. Glover, V. S., President, Ontario Veterinary Association, Ontario Veterinary College, Guelph, Ont.
J. B. Hollingsworth, V. S., Chief Food Inspector, Ottawa, Ont.
Owen McGuirk, V. S., President, Manitoba Veterinary Association, Dauphin, Man.
Wm. Hilton, V. S., Secretary-Treasurer, Manitoba Veterinary Association, Winnipeg, Man.
H. G. Chasmar, V. S., Secretary, Saskatchewan Veterinary Association, Hanley, Sask.
N. A. Johnson, V. A., Secretary, Alberta Veterinary Association, Wetaskiwin, Alta.
T. H. Jagger, B. V. Sc., President, British Columbia Veterinary Association, Vancouver, B. C.
W. Graham Gillam, M. R. C. V. S., Secretary-Treasurer, British Columbia Veterinary Association, Cloverdale, B. C.



Veterinary College at Hanover, Germany.

ASSOCIATION MEETINGS

DELAWARE VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Delaware Veterinary Medical Association was held at Rehoboth Beach, August 28, 1929, with about a dozen members in attendance.

After luncheon at the Hotel Belhaven, the members and guests adjourned to the cottage of Ralph C. Wilson, Secretary of the State Board of Agriculture, where the afternoon session was held.

President F. P. Ruhl, of Milford, made a brief address of welcome, after which Dr. Louis Levinson, who recently attended the meeting of the American Veterinary Medical Association, at Detroit, as official delegate from Delaware, made his report. Dr. Levinson explained the plans that were being formulated for a closer affiliation of the state and national associations and also the recommendations for more uniformity between the various state boards of veterinary medical examiners. He also stated that extensive plans are being made for the International Veterinary Congress, in London, August 4-9, 1930.

The remainder of the meeting was given over to discussion of diseases of especial interest at this time, particularly methods for the control of contagious abortion in cattle and its relation to undulant fever in man, tetanus, hog cholera and tuberculosis eradication.

J. R. PORTEUS, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Academy of Medicine Building, 103rd Street and Fifth Avenue, New York City, October 2, 1929. The meeting was called to order by President H. K. Miller.

Dr. Reginald Blamey was the speaker of the evening, his subject being diseases and surgery of the eye of the dog. He gave a very interesting and thorough talk on the eye, some of the salient points brought forward being as follows:

Always examine the lids thoroughly for turned-in eyelashes. Sometimes we see two rows of eyelashes which turn in and irritate

the corona of the eye. In case the margins of the lids are cut, bring them together with silk sutures. Malposition of the eyes is rather common in Pekes and Chows. It can usually be corrected by cutting the ligament which distorts the eye. In the case of paralysis of the facial nerve, small doses of strychnin sulphate may help. In case of adhesion of the cornea and conjunctiva, anesthetize, separate and cut between adhering surfaces. Be careful of the use of silver solution in the eye because an excessive amount may discolor the cornea. In follicular conjunctivitis, remove the granular excrescences by means of a pair of roller forceps which crush the tissue. In ulcerative keratitis the following prescription is very useful: Antropine sulphate, 2 grs.; cocain hydrochlorid, 1 gr.; solution of adrenalin, 1 gr.; boric acid saturated solution, up to 4 drams.

Dr. Blamey's paper was received with great enthusiasm and a rising vote of thanks was extended him for his contribution.

Dr. Robert S. MacKellar discussed the recent meeting of the American Veterinary Medical Association at Detroit and Dr. J. Elliott Crawford reported the recent New York State Veterinary Medical Society meeting.

There being no further business, motion made and seconded that the meeting be adjourned.

R. J. GARBUTT, *Secretary.*

NORTHEASTERN PENNSYLVANIA VETERINARY MEDICAL CLUB

A meeting of the Northeastern Pennsylvania Veterinary Medical Club was held at the Donovan Hotel, Montrose, Pa., October 11, 1929, at which time Dr. C. J. Marshall, Professor of Veterinary Medicine at the Veterinary Department of the University of Pennsylvania, gave an address on coccidiosis in calves, sweet clover poisoning and hemorrhagic septicemia. An address was also given by Dr. Robert J. Little, of the Pennsylvania Bureau of Animal Industry. Both talks were very interesting and instructive, especially to the busy practitioner.

This was one of the meetings of the Club to which the wives and friends of the veterinarians were invited. The ladies were entertained at card games, while the doctors were in session. About twenty veterinarians attended.

THOS. D. JAMES, *Secretary.*

UTAH VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Utah Veterinary Medical Association was held in the Capitol Building, Salt Lake City, October 11, 1929, with a good percentage of the members in attendance. An interesting program was enjoyed.

One interesting subject was an able paper on anthrax by Dr. N. C. Spalding, giving history, lesions, preventive measures, etc. In the discussion it was stated that in the recent Utah outbreak the use of serum alone was not so effective as when both serum and aggressin were used; also that animals in infected territory should be vaccinated each year before an outbreak occurs. It was stated that a second treatment with serum caused anaphylactic shock.

An outbreak of chicken-pox was reported in Davis County. Dr. W. H. Hendricks, State Veterinarian, reported necrobacillosis in a herd of swine and said two attendants had contracted the infection in their jaws.

Dr. A. C. Johnson, Cedar City, was elected President to succeed himself. Dr. W. H. Hendricks, State Veterinarian, Salt Lake City, was elected Vice-President. Dr. E. A. Bundy, Ogden, was re-elected Secretary-Treasurer.

E. A. Bundy, *Secretary-Treasurer*

ONTARIO VETERINARY ASSOCIATION

The fall meeting of the Ontario Veterinary Association was held at the Ontario Veterinary College, Guelph, October 23-24, 1929. The attendance at the meeting was splendid, upwards of one hundred being present on both days of the session. The President, Dr. J. S. Glover, called the meeting to order at 1:30 p. m. and gave a short address of welcome to those attending. Following this the reports of the Educational Committee, Legislative Committee and Resolutions Committee were heard. At the completion of this business the chairman introduced Dr. A. R. Younie, of St. Catharines, Ontario, who gave an interesting address on "Practical Milk Quality Tests." The speaker handled the subject in a very able manner and distributed statistics regarding some of his work.

The next address was given by Dr. F. W. Schofield, of the Ontario Veterinary College, his subject being "Anemia in Suckling Pigs." The speaker intimated during the course of his remarks that he felt that this condition, so prevalent in pigs now-

adays, was probably due to a disturbance in metabolism resulting from a nutritional or vitamin deficiency. Considerable discussion followed. "Animal Coloration," delivered by Dr. Seymour Hadwen, of the Ontario Research Foundation, was the next item on the program. The substance of the paper was the result of considerable investigational and research work which Dr. Hadwen had conducted while associated with the University of Saskatchewan. The address was very interesting and revealed to those present the ability of the speaker.

The evening session followed a banquet, at which W. Bert Roadhouse, Deputy Minister of Agriculture for Ontario, A. E. Cameron, Chief Veterinary Inspector, Ottawa, and Dr. H. B. Speakman, Director of the Ontario Research Foundation, were present. The first speaker was Dr. Speakman, who outlined the proposed program and function of the Ontario Research Foundation. The importance of the researches in connection with animal diseases was stressed by Dr. Speakman. He intimated the program which had been outlined by the Foundation for the investigation in relation to abortion disease which has already been initiated.

"Abortion Control Measures by the Federal Department of Agriculture" was the subject of an address by Dr. Cameron, in which he outlined requirements of herd-owners wishing to place their herds under the supervision of the Health of Animals Branch. Dr. C. D. McGilvray, Principal of the College, replied to the address of Dr. Speakman in a very appropriate manner and the last speaker of the evening was Mr. Roadhouse, whose remarks were chiefly of a complimentary character. The program of the evening was further enhanced by a number of musical items given between the addresses.

The second day of the meeting was devoted entirely to clinical demonstrations in the surgical amphitheatre of the college. Dr. W. J. R. Fowler had charge of the equine portion of the clinic, Dr. R. A. McIntosh of the cattle clinic, and Dr. J. A. Campbell, of Toronto, of the small-animal clinic. These demonstrators were assisted by a number of other members of the Association. At the conclusion of the clinic the members present expressed themselves as being highly pleased with its character and that much benefit had been derived therefrom.

R. A. McIntosh, *A. V. M. A. Res. Sec. for Ontario*

PENNSYLVANIA STATE VETERINARY MEDICAL ASSOCIATION

The forty-seventh annual meeting of the Pennsylvania State Veterinary Medical Association was held at the Fort Pitt Hotel, Pittsburgh, October 24-25, 1929. The meeting was very well attended by veterinarians from all parts of Pennsylvania, as well as West Virginia and Ohio. Dr. Thomas D. James, of Scranton, presided during the sessions.

The following literary program was presented:

"Undulant Fever and Its Relation to Bang Disease in the Bovine Animal," Dr. Richard A. Kern, Philadelphia.

"Bang Disease Control Work in Fourteen State Institutions," Drs. B. Scott Fritz and M. F. Barnes, Harrisburg.

"Equine Surgical Review," Dr. H. E. Bemis, Philadelphia.

"A Day in a Small-Animal Practice," Dr. R. M. Staley, Narberth.

"Investigations of Canine Diseases with Special References to Rabies—Preliminary Report," Drs. M. F. Barnes and A. N. Metcalf, Harrisburg, and Dr. W. J. Lentz, Philadelphia.

"Feeds and Feeding," Dr. Carl W. Gay, Columbus, Ohio.

"Municipal Milk and Meat Inspection," Dr. H. B. Mitchell, Lancaster.

Reports were received from the delegates of a number of the local veterinary clubs throughout the State, there being thirteen of these holding meetings at stated intervals—some monthly, some bimonthly, others quarterly and a few semiannually.

Among the various committee reports was one from a special committee appointed to study the proposal for affiliating state associations with the American Veterinary Medical Association. The report was made by Dr. C. J. Marshall, chairman of the Committee.

Honorary membership in the Association was conferred upon Dr. Richard A. Kern, of Philadelphia, for his splendid presentation of undulant fever. Dr. Kern has been making an intensive study of the disease in connection with his practice in Philadelphia. Dr. C. H. Stange, of Ames, Iowa, chairman of the Executive Board of the American Veterinary Medical Association, was present and also spoke on investigations of undulant fever being conducted in Iowa.

The election of officers resulted as follows: President, Dr. C. W. Springer, Uniontown; first vice-president, Dr. G. A. Dick, Philadelphia; second vice-president, Dr. E. S. Pickup, Union City; third vice-president, Dr. H. W. Barnard, Lancaster; recording secretary, Dr. B. Scott Fritz, Harrisburg; corresponding secretary, Dr. H. R. Church, Harrisburg; treasurer, Dr. F. U.

Fernsler, Lebanon; trustees, Drs. Thomas D. James, Scranton, H. B. Mitchell, Lancaster, William H. Ivens, Philadelphia, M. B. Herron, Canonsburg, and H. B. Prothero, Johnstown.

The Association adopted a resolution endorsing legislation that would prohibit the cropping of dogs' ears in Pennsylvania.

CENTRAL MICHIGAN VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Central Michigan Veterinary Medical Association was held at the Otsego Hotel, Jackson, October 30, 1929. The meeting was somewhat out of the ordinary in that the entire program was devoted to a discussion of various phases of the abortion problem. The subject was introduced by Dr. E. T. Hallman, of Michigan State College, who discussed the results of his researches, with particular reference to the bacteriological examination of commercial vaccines for infectious abortion. (See paper by Torrey and Hallman to be published in the January issue of the JOURNAL.)

The control of Bang disease from the standpoint of a state official was discussed by Dr. B. J. Killham, of Lansing, State Veterinarian. The relation of the veterinarian to his client in connection with Bang disease control was discussed by Dr. H. Preston Hoskins, Secretary-Editor of the American Veterinary Medical Association. Most of the practitioners who were in attendance discussed the problem from various angles.

An election of officers resulted in Dr. H. M. McConnell, of Litchfield, being chosen for President for the ensuing year and Dr. W. N. Armstrong, of Concord, was re-elected Secretary for his thirteenth consecutive term. The meeting was concluded with a banquet served to about twenty-five veterinarians, a number of whom were accompanied by their wives. A separate entertainment program had been arranged for the ladies in attendance.

California Veterinary Conference

An unusual feature of the California Veterinary Conference, to be held at University Farm, Davis, January 6-10, 1930, will be a series of surgical demonstrations by members of the staff of the Division of Experimental Surgery of the University of California. The operations will be conducted on dogs and cats.

NECROLOGY

GEORGE H. PARKINSON

Dr. George H. Parkinson, of Middletown, Conn., died at Portland, Conn., September 19, 1929, at the age of 72. He had suffered from rheumatism and for the past three years had been confined to his bed a great part of the time.

Dr. Parkinson was a graduate of the Columbia Veterinary College, class of 1881, and joined the A. V. M. A. in 1899.

ALONZO SHECK SHEALY

Dr. Alonzo S. Shealy, of Augusta, Ga., died at a local hospital, October 24, 1929, after an illness dating back about three weeks. He was 56 years old.

After attending Clemson College, South Carolina, Dr. Shealy secured his veterinary training at Iowa State College. He was graduated in 1903 and returned to Clemson College. The following year he went to the Philippines with the Department of Agriculture at Manila. Later he was connected with the Bureau of Science, in Manila, and the College of Veterinary Science, at Los Banos. In 1925, he returned to the United States and located at Augusta, Ga., where he was assigned to duties with Richmond County.

Dr. Shealy joined the A. V. M. A. in 1904. He was a thirty-second degree Scottish Rite Mason and a Shriner. He is survived by his widow and his father.

CHARLES RUSSELL GUILE

Dr. Charles R. Guile, of Canton, N. Y., died at the home of his parents, at Fulton, N. Y., September 13, 1929, after a protracted illness.

Born at Fulton, N. Y., March 20, 1889, Dr. Guile received his early education in the local public schools. Upon finishing high school he entered the New York State Veterinary College at Cornell University. He was graduated in 1913 and located at Canton, N. Y., for general practice.

Dr. Guile joined the A. V. M. A. in 1927. He was a member of the New York State Veterinary Medical Society and Alpha Psi

Fraternity (Beta Chapter). He was a member of the Canton Club and served as its president. He was a member of several Masonic bodies and past grand officer of the Order of Eastern Star. Dr. Guile leaves two sons, his parents, one brother and three sisters.

CHARLES DOBBS RICE

Dr. C. D. Rice, of Ames, Iowa, died November 18, 1929, after an illness of six weeks, due to pyemia.

Born in Richmond, Ky., March 16, 1881, Dr. Rice was graduated from the Richmond High School in 1898 and entered Georgetown College (Ky.). He received the degree of B. S. in 1902. He obtained his veterinary training at Iowa State College, receiving his D. V. M. in 1913. For the two years following graduation he served on the extension staff of Iowa State College, on hog cholera control. From 1915 to 1917 he was employed in commercial work. In the latter year he returned to his Alma Mater, as a member of the staff of the Veterinary Division. At the time of his death he was Associate Professor of Pathology.

Dr. Rice joined the A. V. M. A. in 1916.

GEORGE DEWEY HUSTON

Dr. George D. Huston, of Menden, Kansas, died at a hospital in Topeka, Kansas, August 6, 1929, of Addison's disease. He was a graduate of the Kansas State Agricultural College, class of 1928. He is survived by his widow, mother, two children and three brothers.

Our sympathy goes out to Dr. Charles Massinger, of Phoenixville, Pa., in the death of his wife, Metta W. Massinger, on November 2, 1929.

PERSONALS

BIRTH

To Dr. and Mrs. Channing R. Blatchford, of Flint, Mich., a daughter, Elizabeth Frances, November 7, 1929.

PERSONALS

Dr. B. H. Branson (Ind. '11) has located for general practice in Rosedale, Ind.

Dr. William Caslick (Corn. '27) has removed from Port Lyden, N. Y., to Whitehall, N. Y.

Dr. D. K. Collins (O. S. U. '26) is assisting Dr. Leonard I. Beller (St. Jos. '18), of Lynwood, Calif.

Dr. J. S. Fulton (McK. '18) is veterinary pathologist at the University of Saskatchewan, Saskatoon, Sask.

Dr. C. A. McKillip (McK. '09) has requested a change of address from Rockford, Ill., to Highland Park, Ill.

Dr. J. P. Clark (K. C. V. C. '18), formerly of Pleasant Hill, Mo., has located for general practice at Stronghurst, Ill.

Dr. W. E. Russell (Chi. '20), formerly located at North East, Pa., is now at Northam, Prince Edward Island, Canada.

Dr. J. T. Brown (K. C. V. C. '15) has been reappointed as St. Clair County (Ill.) Veterinarian at a salary of $3600 a year.

Dr. H. A. Salt (O. S. U. '11), of New Philadelphia, Ohio, has been reappointed Tuscarawas County (Ohio) Sanitary Officer.

Dr. A. A. Goodman (Colo. '15), formerly of Norwood, Colo., is now located in the Commercial Club Building, Monte Vista, Colo.

Dr. Henry W. Turner (U. P. '93), of New Hope, Pa., has been elected President of the Bucks County (Pa.) Fair Association.

Dr. Charles B. Eastman (K. C. V. C. '04), formerly in general practice at San Luis Obispo, Calif., is now engaged in public health work in the Dairy Division of the Los Angeles County Health Department.

Dr. C. R. Covington (A. P. I. '21), of the field staff of the Live Stock Sanitary Commission of Texas, is now located at Plainview, Texas.

Dr. E. C. Hughes (Ind. '16), who for the past three years has been Macoupin County (Ill.) Veterinarian, has resumed private practice at Carlinville, Ill.

Dr. C. R. Blatchford (Mich. '26), formerly of Brighton, Mich., is now in Flint, Mich., where he is associated in general practice with Dr. A. E. George (Mich. '20).

Dr. F. W. High (Ont. '14) has been appointed Tuscola County (Mich.) Veterinarian by the Board of Supervisors and will make his headquarters in Caro, Mich.

Dr. E. A. Schmoker (K. S. A. C. '17), formerly of Everett, Wash., is now conducting the Western Veterinary Hospital, 89 and Woodland Park Ave., Seattle, Wash.

Dr. A. H. DeGroot (Gr. Rap. '17), of Dundee, Mich., has been appointed Monroe County (Mich.) Veterinarian, succeeding Dr. S. G. Colby (Mich. '19), of Monroe, Mich.

Dr. Russell B. Booth (Corn. '27) recently announced the removal of his Dog and Cat Hospital from 148-15 Hillside Avenue to 176th Street and Hillside Avenue, Jamaica, N. Y.

Dr. A. A. Moore (Gr. Rap. '12), of Edwardsville, Ill., has been appointed a special agent of the Illinois Department of Agriculture to investigate outbreaks of rabies in the State.

Dr. Horace A. Mills (K. S. A. C. '27), of the New Jersey Department of Agriculture, is now stationed at Jersey City Stock Yards, Jersey City, N. J. He was formerly located in Paterson, N. J.

Dr. H. P. Miller (O. S. U. '97), of Sunbury, Ohio, was recntly honored by being selected as one of ten of Ohio's outstanding farmers and rural leaders selected for the honorary degree of Master Farmer.

Dr. G. W. Musselman (McK. '11), of Denver, Ind., has started the construction of a new veterinary hospital and office building to take the place of the one which was destroyed by fire several months ago.

Dr. C. W. Olson (Ont. '22) has resigned his position in the Pathological Division, Bureau of Animal Industry, Washington, D. C., and has located at New Ulm, Minn., where he will engage in general practice.

Dr. C. B. Teel (Ind. '15), of Arcola, Ill., has been appointed veterinarian to the State Department of Agriculture, in connection with tuberculosis eradication work. He has been assigned to the Chicago Stock Yards.

Dr. S. J. Schilling (O. S. U. '17) has resigned his position in the Department of Animal Industry, Alabama Polytechnic Institute, to join the staff of the Jensen-Salsbery Laboratories, at Kansas City, Mo., December 1.

Dr. Paul Woods (Chi. '03), of Greenville, Ill., has been appointed Macoupin County Veterinarian by the Finance Committee of the Board of Supervisors, succeeding Dr. G. B. Hughes who has held this position for the past two years.

Dr. W. A. Axby (Cin. '05-Ohio '95), of Harrison, Ohio, was recently honored with the unanimous election to the post of Lieutenant Governor of the Third Ohio Division in Kiwanis, at the convention held recently at Marietta, Ohio.

Dr. E. F. Sanders (K. S. A. C. '27), who has been at the Massachusetts Agricultural College, Amherst, for some time, has resigned to accept a position in the Department of Veterinary Science, University of Missouri, at Columbia.

Dr. Louis A. Klein (U. P. '97), Dean of the School of Veterinary Medicine, University of Pennsylvania, has been reappointed a member of the Committee on Standards and Methods of the American Association of Medical Milk Commissions.

Dr. E. M. Alderman (K. C. V. C. '14) has been transferred from swine sanitation and hog cholera control work, with headquarters at Yazoo City, Miss., to contagious abortion work, at Jackson, Miss., with the State Live Stock Sanitary Board.

Dr. G. T. Woodward (Wash. '24), of Fallon, Nevada, has been appointed a member of the Nevada State Board of Veterinary Medical Examiners, by Governor Balzar, succeeding Dr. Robert Dill (K. C. V. C. '04), of Reno, Nevada, resigned.

Dr. Frank L. Whitcomb (Wash. '29), of Crary, N. Dak., was the only applicant for a license to practice in North Dakota, at the meeting of the State Veterinary Medical Examining Board, in Bismarck, on October 9, 1929. Dr. Whitcomb was successful.

Dr. John B. Mohler (U. P. '96), Chief, U. S. Bureau of Animal Industry, represented the Department of Agriculture and presided, at the request of those present, at a trade practice conference for the meat packing and wholesale meat industry, held at the invitation of Hon. Arthur M. Hyde, Secretary of Agriculture, at Chicago, Illinois, October 22, 1929.